APPENDIX D

MTCA *Stat* v.2.1 Table
Systat v.9 Tables

APPENDIX D
MTCA *Stat* v.2.1 Tables
Systat v.9 Tables

MTCA *Stat* v.2.1 was used in this risk assessment to determine the distribution of the data, because the formula used to calculate the UCL_{95} depends on the data distribution. The MTCA *Stat* v.2.1 results indicated either a lognormal distribution or rejected both the normal and lognormal distributions. Therefore, the bootstrap method, a nonparametric statistical technique, was used to calculate the UCL_{95} using Systat v.9. See Section 6 for a detailed discussion.

1.2   River Road 95
2.7   Antimony
2.8
2.2
4.1
1.6
3.7

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	2.61
Censored		Lognormal mean	2.67
Detection limit or PQL		Std. devn.	1.051077
Method detection limit		Median	2.7
TOTAL	7	Min.	1.2
		Max.	4.1

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.954. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 3.39

21.4   River Road 95
23.4   Arsenic
28.4
21.6
35.1
21.8
31.7

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	26.20	
Censored		Lognormal mean	26.27	
Detection limit or PQL		Std. devn.	5.563272	
Method detection limit		Median	23.4	
TOTAL	7	Min.	21.4	
		Max.	35.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8502. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 30.29

10.1   River Road 95
14.5   Cadmium
18
16.2
17.4
11
21

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	15.46	
Censored		Lognormal mean	15.56	
Detection limit or PQL		Std. devn.	3.894379	
Method detection limit		Median	16.2	
TOTAL	7	Min.	10.1	
		Max.	21	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.93. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 18.32

27600   River Road 95
25200   Iron
27500
23300
28000
25400
27200

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	26314.29	
Censored		Lognormal mean	26323.59	
Detection limit or PQL		Std. devn.	1724.75	
Method detection limit		Median	27200	
TOTAL	7	Min.	23300	
		Max.	28000	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8682. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 27580.92

719
1310
2360
656
2350
867
1590

River Road 95

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	1407.43
Censored		Lognormal mean	1440.45
Detection limit or PQL		Std. devn.	726.7851
Method detection limit		Median	1310
TOTAL	7	Min.	656
		Max.	2360

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8978. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1941.17

1650
1880
2820
1740
2890
1710
2810

River Road 95

Manganese

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	2214.29
Censored			Lognormal mean	2224.73
Detection limit or PQL			Std. devn.	589.883
Method detection limit			Median	1880
TOTAL	7		Min.	1650
			Max.	2890

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7722. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.7595. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 2647.49

0.15
0.23
0.28
0.22
0.45
0.16
0.55

River Road 95

Mercury

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.29
Censored		Lognormal mean	0.30
Detection limit or PQL		Std. devn.	0.151814
Method detection limit		Median	0.23
TOTAL	7	Min.	0.15
		Max.	0.55

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9242. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.4

2190   River Road 95
2420   Zinc
3230
2040
3320
2490
3270

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	2708.57
Censored		Lognormal mean	2716.94
Detection limit or PQL		Std. devn.	548.921
Method detection limit		Median	2490
TOTAL	7	Min.	2040
		Max.	3320

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8634. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 3111.69

0.5   Harvard Road N
0.5   Antimony
1.9
1.5
1.6
3.1
2

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	1.59	
Censored		Lognormal mean	1.69	
Detection limit or PQL		Std. devn.	0.906327	
Method detection limit		Median	1.6	
TOTAL	7	Min.	0.5	
		Max.	3.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8511. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 2.25

15.3   Harvard Road N
15.1   Arsenic
21.6
17
15.8
23.6
19.1

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	18.21
Censored			Lognormal mean	18.25
Detection limit or PQL			Std. devn.	3.33538
Method detection limit			Median	17
TOTAL	7		Min.	15.1
			Max.	23.6

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8932. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 20.66

9.3
6.4
9.9
9.1
10.1
13.6
7

Harvard Road N

Cadmium

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	9.34
Censored		Lognormal mean	9.38
Detection limit or PQL		Std. devn.	2.352911
Method detection limit		Median	9.3
TOTAL	7	Min.	6.4
		Max.	13.6

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9431. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 11.07

23700
24400
29800
27000
28100
29000
30400

Harvard Road N

Iron

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	27485.71	
Censored		Lognormal mean	27505.94	
Detection limit or PQL		Std. devn.	2601.556	
Method detection limit		Median	28100	
TOTAL	7	Min.	23700	
		Max.	30400	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9042. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 29396.26

479　　　　Harvard Road N
328　　　　Lead
484
379
503
534
261

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	424.00
Censored		Lognormal mean	426.95
Detection limit or PQL		Std. devn.	102.2872
Method detection limit		Median	479
TOTAL	7	Min.	261
		Max.	534

Lognormal distribution?　　　　Normal distribution?
r-squared is:　　　　　　　　　r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8797. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 499.12

1520   Harvard Road N
1260   Manganese
1970
1470
944
1270
970

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	1343.43
Censored		Lognormal mean	1349.57
Detection limit or PQL		Std. devn.	353.8205
Method detection limit		Median	1270
TOTAL	7	Min.	944
		Max.	1970

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.945. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1603.27

0.18
0.17
0.28
0.18
0.18
0.29
0.18

Harvard Road N

Mercury

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.21	
Censored		Lognormal mean	0.21	
Detection limit or PQL		Std. devn.	0.052418	
Method detection limit		Median	0.18	
TOTAL	7	Min.	0.17	
		Max.	0.29	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6812. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6672. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.25

2020
1880
2340
2090
2140
2480
1430

Harvard Road N

Zinc

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	2054.29
Censored		Lognormal mean	2060.54
Detection limit or PQL		Std. devn.	339.7969
Method detection limit		Median	2090
TOTAL	7	Min.	1430
		Max.	2480

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9056. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 2303.83

2   Harvard Road S
0.86   Antimony
1.1
0.67

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	4	Mean	1.16	
Censored		Lognormal mean	1.18	
Detection limit or PQL		Std. devn.	0.588579	
Method detection limit		Median	0.98	
TOTAL	4	Min.	0.67	
		Max.	2	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9544. This exceeds the tabled value of 0.748

UCL (based on t-statistic) is 1.85

31.7   Harvard Road S
16.2   Arsenic
13.9
13.2
16.4
13.5
13.6

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	16.93	
Censored		Lognormal mean	16.93	
Detection limit or PQL		Std. devn.	6.644726	
Method detection limit		Median	13.9	
TOTAL	7	Min.	13.2	
		Max.	31.7	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6834. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.616. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 21.81

11.4   Harvard Road S
4.1   Cadmium
4
4.2
7.8
5.7
5.3

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	6.07
Censored		Lognormal mean	6.10
Detection limit or PQL		Std. devn.	2.701675
Method detection limit		Median	5.3
TOTAL	7	Min.	4
		Max.	11.4

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8705. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 8.06

25700   Harvard Road S
21100   Iron
21600
21000
22200
20100
19800

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	21642.86
Censored			Lognormal mean	21652.19
Detection limit or PQL			Std. devn.	1968.804
Method detection limit			Median	21100
TOTAL	7		Min.	19800
			Max.	25700

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8593. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 23088.72

1070　　　　Harvard Road S.
234　　　　Lead
146
154
306
326
335

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	367.29
Censored			Lognormal mean	366.43
Detection limit or PQL			Std. devn.	319.5135
Method detection limit			Median	306
TOTAL	7		Min.	146
			Max.	1070

Lognormal distribution?　　　　Normal distribution?
r-squared is:　　　　　　　　　r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8731. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 601.93

2850
1150
 879
 882
1180
 978
1110

Harvard Road S

Manganese

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	1289.86
Censored			Lognormal mean	1285.29
Detection limit or PQL			Std. devn.	698.8596
Method detection limit			Median	1110
TOTAL	7		Min.	879
			Max.	2850

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7207. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6154. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 1803.09

0.24
0.06
0.025
0.025
0.08
0.06
0.06

Harvard Road S

Mercury

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.08	
Censored		Lognormal mean	0.08	
Detection limit or PQL		Std. devn.	0.073977	
Method detection limit		Median	0.06	
TOTAL	7	Min.	0.025	
		Max.	0.24	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8782. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.13

2640   Harvard Road S
2180   Zinc
1570
1180
1770
1360
1500

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	1742.86	
Censored		Lognormal mean	1750.66	
Detection limit or PQL		Std. devn.	508.0917	
Method detection limit		Median	1570	
TOTAL	7	Min.	1180	
		Max.	2640	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9668. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 2115.99

0.5
1.9
3
2.5
2.8
2.9
2

Barker Road N

Antimony

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	2.23	
Censored		Lognormal mean	2.41	
Detection limit or PQL		Std. devn.	0.875051	
Method detection limit		Median	2.5	
TOTAL	7	Min.	0.5	
		Max.	3	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.699. This is less than the tabled value of 0.803
Assume normal distribution.
W value is 0.8477. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 2.87

13
22.3
29.8
28.5
30.8
43.8
45.6

Barker Road N

Arsenic

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	30.54	
Censored		Lognormal mean	31.17	
Detection limit or PQL		Std. devn.	11.42335	
Method detection limit		Median	29.8	
TOTAL	7	Min.	13	
		Max.	45.6	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9133. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 38.93

3.5 Barker Road N
6.5 Cadmium
15.5
12.6
13.3
13
11.1

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	10.79
Censored			Lognormal mean	11.25
Detection limit or PQL			Std. devn.	4.248305
Method detection limit			Median	12.6
TOTAL	7		Min.	3.5
			Max.	15.5

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8051. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 13.91

26100
38100
29900
31900
33600
49300
43800

Barker Road N

Iron

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	36100.00	
Censored		Lognormal mean	36218.57	
Detection limit or PQL		Std. devn.	8163.537	
Method detection limit		Median	33600	
TOTAL	7	Min.	26100	
		Max.	49300	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9764. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 42095.18

116
106
822
647
714
537
404

Barker Road N.
Lead

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	478.00
Censored		Lognormal mean	540.45
Detection limit or PQL		Std. devn.	283.1307
Method detection limit		Median	537
TOTAL	7	Min.	106
		Max.	822

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8083. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 685.93

687
909
1720
1480
1610
1310
1660

Barker Road N

Manganese

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	1339.43
Censored			Lognormal mean	1358.46
Detection limit or PQL			Std. devn.	398.4068
Method detection limit			Median	1480
TOTAL	7		Min.	687
			Max.	1720

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8368. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1632.01

0.05
0.05
0.38
0.28
0.34
0.18
0.17

Barker Road N

Mercury

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.21
Censored			Lognormal mean	0.23
Detection limit or PQL			Std. devn.	0.131873
Method detection limit			Median	0.18
TOTAL	7		Min.	0.05
			Max.	0.38

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8426. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.3

1990
1360
2590
2610
2490
3470
4880

Barker Road N

Zinc

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	2770.00
Censored		Lognormal mean	2803.35
Detection limit or PQL		Std. devn.	1131.003
Method detection limit		Median	2590
TOTAL	7	Min.	1360
		Max.	4880

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9701. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 3600.59

1.2   N Flora Road
1.4   Antimony
0.66
0.93

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	1.28	
Censored		Lognormal mean	1.30	
Detection limit or PQL		Std. devn.	0.386172	
Method detection limit		Median	1.4	
TOTAL	7	Min.	0.66	
		Max.	1.7	

1.7
1.4
1.7

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8893. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1.57

15.9
16.4
19.8
17.6
22.5
20.3
24.8

N Flora Road

Arsenic

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	19.61
Censored			Lognormal mean	19.65
Detection limit or PQL			Std. devn.	3.263653
Method detection limit			Median	19.8
TOTAL	7		Min.	15.9
			Max.	24.8

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9529. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 22.01

5.4   N Flora Road
5.2   Cadmium
5.5
7.3
10.1
9.4
10.1

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	7.57	
Censored		Lognormal mean	7.63	
Detection limit or PQL		Std. devn.	2.266947	
Method detection limit		Median	7.3	
TOTAL	7	Min.	5.2	
		Max.	10.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8214. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 9.24

24000
27700
26900
25400
25200
27200
28700

N Flora Road

Iron

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	26442.86
Censored			Lognormal mean	26450.38
Detection limit or PQL			Std. devn.	1635.907
Method detection limit			Median	26900
TOTAL	7		Min.	24000
			Max.	28700

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9626. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 27644.24

799   N. Flora Road
529   Lead
771
531
1040
498
772

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	705.71
Censored		Lognormal mean	709.45
Detection limit or PQL		Std. devn.	197.4856
Method detection limit		Median	771
TOTAL	7	Min.	498
		Max.	1040

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8813. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 850.74

1770   N Flora Road
1340   Manganese
1390
1300
2110
1420
1650

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	1568.57	
Censored		Lognormal mean	1571.29	
Detection limit or PQL		Std. devn.	294.0198	
Method detection limit		Median	1420	
TOTAL	7	Min.	1300	
		Max.	2110	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8938. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1784.5

0.11	N Flora Road
0.06	Mercury
0.08	
0.025	
0.16	
0.19	
0.11	

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	0.11
Censored		Lognormal mean	0.11
Detection limit or PQL		Std. devn.	0.056789
Method detection limit		Median	0.11
TOTAL	7	Min.	0.025
		Max.	0.19

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9252. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.15

2440   N Flora Road
4020   Zinc
4450
3860
2930
3030
2990

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	3388.57
Censored		Lognormal mean	3399.81
Detection limit or PQL		Std. devn.	724.0034
Method detection limit		Median	3030
TOTAL	7	Min.	2440
		Max.	4450

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.932. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 3920.27

0.5
1.6
0.5
1.2
0.5
0.5
0.49

Plante Ferry Park

Antimony

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.76
Censored			Lognormal mean	0.76
Detection limit or PQL			Std. devn.	0.455041
Method detection limit			Median	0.5
TOTAL	7		Min.	0.49
			Max.	1.6

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6541. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6579. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 1.09

16.5   Plante Ferry Park
15.2   Arsenic
14.2
14
12.1
5.2
7.6

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	12.11	
Censored		Lognormal mean	12.40	
Detection limit or PQL		Std. devn.	4.180283	
Method detection limit		Median	14	
TOTAL	7	Min.	5.2	
		Max.	16.5	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8291. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 15.18

0.58
1.3
2.5
2.4
0.1
0.1
0.1

Plante Ferry Park
Cadmium

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	1.01
Censored		Lognormal mean	1.45
Detection limit or PQL		Std. devn.	1.072807
Method detection limit		Median	0.58
TOTAL	7	Min.	0.1
		Max.	2.5

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8081. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1.8

27700
42900
23600
24400
28000
13800
20500

Plante Ferry Park

Iron

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	25842.86
Censored			Lognormal mean	26076.35
Detection limit or PQL			Std. devn.	8941.823
Method detection limit			Median	24400
TOTAL	7		Min.	13800
			Max.	42900

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.957. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 32409.6

88.2
73.2
173
174
98.1
33.7
112

Plante Ferry Park

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	107.46	
Censored		Lognormal mean	111.79	
Detection limit or PQL		Std. devn.	51.34283	
Method detection limit		Median	98.1	
TOTAL	7	Min.	33.7	
		Max.	174	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9128. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 145.16

503
286
673
704
645
129
321

Plante Ferry Park

Manganese

	MTCA*Stat* 2.1			
Number of samples			Uncensored values	
Uncensored	7		Mean	465.86
Censored			Lognormal mean	492.31
Detection limit or PQL			Std. devn.	223.5684
Method detection limit			Median	503
TOTAL	7		Min.	129
			Max.	704

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8662. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 630.04

0.18
0.05
0.05
0.05
0.05
0.05
0.05

Plante Ferry Park

Mercury

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.07
Censored			Lognormal mean	0.07
Detection limit or PQL			Std. devn.	0.049135
Method detection limit			Median	0.05
TOTAL	7		Min.	0.05
			Max.	0.18

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4533. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.4556. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.1

266
365
592
614
228
119
254

Plante Ferry Park

Zinc

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	348.29	
Censored		Lognormal mean	359.36	
Detection limit or PQL		Std. devn.	188.4062	
Method detection limit		Median	266	
TOTAL	7	Min.	119	
		Max.	614	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9359. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 486.65

7.7   Boulder Beach
5.5   Arsenic
5.6
6.9
5.1
3.8
3.1

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	5.39
Censored		Lognormal mean	5.44
Detection limit or PQL		Std. devn.	1.608756
Method detection limit		Median	5.5
TOTAL	7	Min.	3.1
		Max.	7.7

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9512. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 6.57

22600
15700
19400
18100
13800
 9470
 8280

Boulder Beach

Iron

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	15335.71	
Censored		Lognormal mean	15545.90	
Detection limit or PQL		Std. devn.	5221.915	
Method detection limit		Median	15700	
TOTAL	7	Min.	8280	
		Max.	22600	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.93. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 19170.61

30.2   Boulder Beach
25.1   Lead
24.8
54.6
40.4
18.1
21.4

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	30.66
Censored		Lognormal mean	30.87
Detection limit or PQL		Std. devn.	12.76034
Method detection limit		Median	25.1
TOTAL	7	Min.	18.1
		Max.	54.6

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9473. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 40.03

507   Boulder Beach

395   Manganese

633

526

416

300

281

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	436.86	
Censored		Lognormal mean	439.97	
Detection limit or PQL		Std. devn.	126.8509	
Method detection limit		Median	416	
TOTAL	7	Min.	281	
		Max.	633	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9495. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 530.01

99.6   Boulder Beach
73.6   Zinc
87.7
 172
82.3
50.5
49.4

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	87.87
Censored			Lognormal mean	88.63
Detection limit or PQL			Std. devn.	41.48613
Method detection limit			Median	82.3
TOTAL	7		Min.	49.4
			Max.	172

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9294. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 118.34

0.49
0.495
0.49
0.49
0.495
0.485
0.5

People's Park

Antimony

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.49
Censored		Lognormal mean	0.49
Detection limit or PQL		Std. devn.	0.00488
Method detection limit		Median	0.49
TOTAL	7	Min.	0.485
		Max.	0.5

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 1.0191. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.5

10.2   People's Park
8.7   Arsenic
11.9
25.2
10.3
8.8
14.6

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	12.81	
Censored		Lognormal mean	12.84	
Detection limit or PQL		Std. devn.	5.825641	
Method detection limit		Median	10.3	
TOTAL	7	Min.	8.7	
		Max.	25.2	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8375. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 17.09

0.1   People's Park
0.1   Cadmium
0.1
0.1

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.10
Censored			Lognormal mean	0.10
Detection limit or PQL			Std. devn.	0.00189
Method detection limit			Median	0.1
TOTAL	7		Min.	0.095
			Max.	0.1

0.1
0.1
0.095
0.1

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4434. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.1

20200
20100
25500
28300
22300
20000
25500

People's Park

Iron

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	23128.57
Censored			Lognormal mean	23159.33
Detection limit or PQL			Std. devn.	3322.005
Method detection limit			Median	22300
TOTAL	7		Min.	20000
			Max.	28300

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8611. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 25568.2

15.4
14.5
18.6
13.4
16
26.6
13.2

People's Park (Latah Creek)

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	16.81	
Censored		Lognormal mean	16.85	
Detection limit or PQL		Std. devn.	4.684879	
Method detection limit		Median	15.4	
TOTAL	7	Min.	13.2	
		Max.	26.6	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8462. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 20.25

331   People's Park
293   Manganese
423
390
469
414
489

MTCA*Stat* 2.1		
Number of samples		Uncensored values
Uncensored	7	Mean 401.29
Censored		Lognormal mean 402.44
Detection limit or PQL		Std. devn. 70.36267
Method detection limit		Median 414
TOTAL	7	Min. 293
		Max. 489

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9394. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 452.96

69.2   People's Park
77.5   Zinc
90.7
78.4

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	85.96	
Censored		Lognormal mean	86.08	
Detection limit or PQL		Std. devn.	25.94333	
Method detection limit		Median	78	
TOTAL	7	Min.	65.9	
		Max.	142	

78
142
65.9

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8016. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.7241. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 105.01

7.1
6.1
7.1
6.5
9.4
8.4
18.2

Riverside Park

Arsenic

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	8.97
Censored		Lognormal mean	8.97
Detection limit or PQL		Std. devn.	4.22363
Method detection limit		Median	7.1
TOTAL	7	Min.	6.1
	Max.		18.2

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7988. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6915. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 12

0.75   Riverside Park
0.36   Cadmium
0.87
2
1.7
2.5
1.5

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	1.38	
Censored		Lognormal mean	1.47	
Detection limit or PQL		Std. devn.	0.758611	
Method detection limit		Median	1.5	
TOTAL	7	Min.	0.36	
	Max.		2.5	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9332. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 1.93

12000
12600
14300
14500
14500
14800
17900

Riverside Park

Iron

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	14371.43
Censored		Lognormal mean	14386.79
Detection limit or PQL		Std. devn.	1890.074
Method detection limit		Median	14500
TOTAL	7	Min.	12000
Max.			17900

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9117. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 15700.18

98
41.4
57.1
110
88.7
92
79.7

Riverside Park

Lead

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	80.99
Censored		Lognormal mean	82.09
Detection limit or PQL		Std. devn.	23.98384
Method detection limit		Median	88.7
TOTAL	7	Min.	41.4
		Max.	110

Lognormal distribution?	Normal distribution?
r-squared is:	r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8823. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 98.6

168   Riverside Park
147   Manganese
132
140
203
258
345

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	199.00	
Censored		Lognormal mean	200.10	
Detection limit or PQL		Std. devn.	77.94015	
Method detection limit		Median	168	
TOTAL	7	Min.	132	
		Max.	345	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9053. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 256.24

0.46   Riverside Park
0.11   Mercury
0.06
0.12
0.06
0.09
0.025

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.13
Censored		Lognormal mean	0.13
Detection limit or PQL		Std. devn.	0.148208
Method detection limit		Median	0.09
TOTAL	7	Min.	0.025
		Max.	0.46

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9367. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.24

230   Riverside Park
169   Zinc
232
436
353
377
337

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	304.86
Censored			Lognormal mean	308.16
Detection limit or PQL			Std. devn.	95.84958
Method detection limit			Median	337
TOTAL	7		Min.	169
			Max.	436

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9241. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 375.25

0.49
0.485
0.5
0.48
0.49
0.55
0.485

Wynecoop Landing
Antimony

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.50	
Censored		Lognormal mean	0.50	
Detection limit or PQL		Std. devn.	0.024128	
Method detection limit		Median	0.49	
TOTAL	7	Min.	0.48	
		Max.	0.55	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Reject lognormal distribution.
W value is 0.6942. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6871. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.51

10.2   Wynecoop Landing
10
9
9.2
10.2
11.5
10.1

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	10.03
Censored		Lognormal mean	10.03
Detection limit or PQL		Std. devn.	0.813868
Method detection limit		Median	10.1
TOTAL	7	Min.	9
		Max.	11.5

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9072. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 10.63

0.1
0.1
0.1
0.095
0.1
0.1
0.095

Wynecoop Landing

Cadmium

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.10
Censored			Lognormal mean	0.10
Detection limit or PQL			Std. devn.	0.00244
Method detection limit			Median	0.1
TOTAL	7		Min.	0.095
			Max.	0.1

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6119. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.1

17400
19600
20200
20300
19800
22300
20800

Wynecoop Landing

Iron

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	20057.14
Censored			Lognormal mean	20065.84
Detection limit or PQL			Std. devn.	1471.798
Method detection limit			Median	20200
TOTAL	7		Min.	17400
			Max.	22300

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9224. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 21138.01

14.6
17.2
15.8
15.3
16.1
15.7
16.3

Wynecoop Landing

Lead

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	15.86
Censored		Lognormal mean	15.86
Detection limit or PQL		Std. devn.	0.81416
Method detection limit		Median	15.8
TOTAL	7	Min.	14.6
		Max.	17.2

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9872. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 16.46

351   Wynecoop Landing
392   Manganese
387
443   MTCA*Stat* 2.1
439   Number of samples   Uncensored values
552   Uncensored   7   Mean   438.43
505   Censored   Lognormal mean   439.16
           Detection limit or PQL   Std. devn.   70.46951
           Method detection limit   Median   439
                TOTAL   7   Min.   351
                                               Max.   552

           Lognormal distribution?   Normal distribution?
           r-squared is: r-squared is:
           Recommendations:
           Assume lognormal distribution.
           W value is 0.9633. This exceeds the tabled value of 0.803

                        UCL (based on t-statistic) is 490.18

0.18   Wynecoop Landing
0.35   Mercury
0.05
0.05
0.05
0.05
0.05

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	0.11
Censored		Lognormal mean	0.11
Detection limit or PQL		Std. devn.	0.11582
Method detection limit		Median	0.05
TOTAL	7	Min.	0.05
		Max.	0.35

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6507. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6374. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.2

88.8   Wynecoop Landing
146   Zinc
121
112
95.3
88.1
88.1

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	105.61
Censored		Lognormal mean	105.83
Detection limit or PQL		Std. devn.	22.02691
Method detection limit		Median	95.3
TOTAL	7	Min.	88.1
		Max.	146

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8511. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 121.79

8.3   Coyote Spit
9.8   Arsenic
9.9
6.5
10.4
9.6
9.2

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	9.10	
Censored		Lognormal mean	9.12	
Detection limit or PQL		Std. devn.	1.321615	
Method detection limit		Median	9.6	
TOTAL	7	Min.	6.5	
		Max.	10.4	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8177. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 10.07

0.05
0.05
0.05
0.27
0.05
0.05
0.05

Coyote S[it

Cadmium

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.08	
Censored		Lognormal mean	0.08	
Detection limit or PQL		Std. devn.	0.083152	
Method detection limit		Median	0.05	
TOTAL	7	Min.	0.05	
		Max.	0.27	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Reject lognormal distribution.
W value is 0.4533. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.4532. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.14

17700   Coyote Spit
19500   Iron
18600
16800
20200
19300
18800

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	18700.00	
Censored		Lognormal mean	18705.50	
Detection limit or PQL		Std. devn.	1146.008	
Method detection limit		Median	18800	
TOTAL	7	Min.	16800	
		Max.	20200	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9594. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 19541.61

18.2
19.2
19.9
19.9
25.1
16.7
20

Coyote Spit

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	19.86	
Censored		Lognormal mean	19.87	
Detection limit or PQL		Std. devn.	2.603112	
Method detection limit		Median	19.9	
TOTAL	7	Min.	16.7	
		Max.	25.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.886. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 21.77

293   Coyote Spit
321   Manganese
268
229
318
241
267

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	276.71
Censored		Lognormal mean	277.06
Detection limit or PQL		Std. devn.	35.71514
Method detection limit		Median	268
TOTAL	7	Min.	229
		Max.	321

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.932. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 302.94

0.035
0.025
0.025
0.025
0.025
0.025
0.025

Coyote Spit

Mercury

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.03	
Censored		Lognormal mean	0.03	
Detection limit or PQL		Std. devn.	0.00378	
Method detection limit		Median	0.025	
TOTAL	7	Min.	0.025	
		Max.	0.035	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4534. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.03

92.9
147
172
282
298
101
202

Coyote Spit

Zinc

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	184.99	
Censored		Lognormal mean	188.29	
Detection limit or PQL		Std. devn.	81.25926	
Method detection limit		Median	172	
TOTAL	7	Min.	92.9	
		Max.	298	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9308. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 244.66

7.3   The Docks
7.8   Arsenic
6.9
7.3
13.3
7
9.4

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	8.43	
Censored		Lognormal mean	8.44	
Detection limit or PQL		Std. devn.	2.309195	
Method detection limit		Median	7.3	
TOTAL	7	Min.	6.9	
		Max.	13.3	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7649. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.7161. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 10.12

0.05 The Docks
0.05 Cadmium
0.05
0.05
0.05
0.24
0.05

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.08
Censored			Lognormal mean	0.07
Detection limit or PQL			Std. devn.	0.071813
Method detection limit			Median	0.05
TOTAL	7		Min.	0.05
			Max.	0.24

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4532. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.4524. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.13

25400   The Docks
27400   Iron
24700
25300
24200
22300
25300

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	24942.86
Censored			Lognormal mean	24950.00
Detection limit or PQL			Std. devn.	1532.816
Method detection limit			Median	25300
TOTAL	7		Min.	22300
			Max.	27400

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9265. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 26068.53

18.2   The Docks
17.7   Lead
16.6
19.4
16.7
23.5
17.9

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	18.57
Censored		Lognormal mean	18.58
Detection limit or PQL		Std. devn.	2.370453
Method detection limit		Median	17.9
TOTAL	7	Min.	16.6
		Max.	23.5

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.832. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 20.31

272   The Docks
289   Manganese
255
319
315
436
419

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	329.29
Censored		Lognormal mean	330.12
Detection limit or PQL		Std. devn.	70.91007
Method detection limit		Median	315
TOTAL	7	Min.	255
		Max.	436

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8959. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 381.36

0.025   The Docks
0.025   Mercury
0.025
0.025
0.025
0.025
0.025

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.03	
Censored		Lognormal mean	0.03	
Detection limit or PQL		Std. devn.	3.8E-10	
Method detection limit		Median	0.025	
TOTAL	7	Min.	0.025	
		Max.	0.025	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.03

86.8   The Docks
93.7   Zinc
72.5
142
80.6
265
76.8

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	116.77	
Censored		Lognormal mean	116.63	
Detection limit or PQL		Std. devn.	69.40799	
Method detection limit		Median	86.8	
TOTAL	7	Min.	72.5	
		Max.	265	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7916. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.6935. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 167.74

0.5   Jackson Cove
0.5   Antimony
0.5
0.5

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.59	
Censored		Lognormal mean	0.59	
Detection limit or PQL		Std. devn.	0.226779	
Method detection limit		Median	0.5	
TOTAL	7	Min.	0.5	
		Max.	1.1	

1.1   (values in left margin: 0.5, 0.5, 0.5, 0.5, 1.1, 0.5, 0.5)

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4532. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.4534. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.75

10.1   Jackson Cove
9.1   Arsenic
10.9
13.2
13.3
11.7
22.9

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	13.03
Censored			Lognormal mean	13.06
Detection limit or PQL			Std. devn.	4.616172
Method detection limit			Median	11.7
TOTAL	7		Min.	9.1
			Max.	22.9

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8659. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 16.42

0.1   Jackson Cove
0.1   Cadmium
0.1
0.1

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	0.10
Censored		Lognormal mean	0.10
Detection limit or PQL		Std. devn.	1.52E-09
Method detection limit		Median	0.1
TOTAL	7	Min.	0.1
		Max.	0.1

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.1

23500   Jackson Cove
23100   Iron
24300
27500
26900
25500
22800

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	24800.00	
Censored		Lognormal mean	24809.45	
Detection limit or PQL		Std. devn.	1871.719	
Method detection limit		Median	24300	
TOTAL	7	Min.	22800	
		Max.	27500	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9089. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 26174.56

13.8
18.4
14.1
14.1
12.5
13.6
20

Jackson Cove

Lead

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	15.21
Censored			Lognormal mean	15.24
Detection limit or PQL			Std. devn.	2.813911
Method detection limit			Median	14.1
TOTAL	7		Min.	12.5
			Max.	20

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8255. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 17.28

288
387
393
468
428
543
531

Jackson Cove

Manganese

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	434.00	
Censored		Lognormal mean	435.85	
Detection limit or PQL		Std. devn.	89.15156	
Method detection limit		Median	428	
TOTAL	7	Min.	288	
		Max.	543	

Lognormal distribution?　　　　Normal distribution?
r-squared is:　　　　　　　　　　r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9321. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 499.47

0.05
0.05
0.05
0.05   Jackson Cove
0.05   Mercury
0.05
0.05

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.05	
Censored		Lognormal mean	0.05	
Detection limit or PQL		Std. devn.	7.6E-10	
Method detection limit		Median	0.05	
TOTAL	7	Min.	0.05	
		Max.	0.05	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.05

51.3   Jackson Cove

147   Zinc

124

70.4

70

96.1

207

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	109.40
Censored		Lognormal mean	111.18
Detection limit or PQL		Std. devn.	54.43427
Method detection limit		Median	96.1
TOTAL	7	Min.	51.3
		Max.	207

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9698. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 149.38

8.5
9.9
6.6
9
11.3
13
8.2

Porcupine Bay

Arsenic

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	9.50	
Censored		Lognormal mean	9.53	
Detection limit or PQL		Std. devn.	2.12132	
Method detection limit		Median	9	
TOTAL	7	Min.	6.6	
		Max.	13	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.987. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 11.06

0.05
0.045
0.05
0.05
0.05
0.05
0.05

Porcupine Bay

Cadmium

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.05
Censored			Lognormal mean	0.05
Detection limit or PQL			Std. devn.	0.00189
Method detection limit			Median	0.05
TOTAL	7		Min.	0.045
			Max.	0.05

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4519. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.05

13400   Porcupine Bay
13900   Iron
12400
13800
19000
19000
13700

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	15028.57	
Censored		Lognormal mean	15053.40	
Detection limit or PQL		Std. devn.	2758.45	
Method detection limit		Median	13800	
TOTAL	7	Min.	12400	
		Max.	19000	

Lognormal distribution?   Normal distribution?
r-squared is: 0.784   r-squared is: 0.759

Recommendations:
Reject lognormal distribution.
W value is 0.7734. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.7468. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 17054.34

11
14.4
13.4
11.5
16.9
20.2
15.9

Porcupine Bay

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	14.76	
Censored		Lognormal mean	14.80	
Detection limit or PQL		Std. devn.	3.220174	
Method detection limit		Median	14.4	
TOTAL	7	Min.	11	
		Max.	20.2	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9703. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 17.12

204   Porcupine Bay
221   Manganese
196
231
361
601
187

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	285.86
Censored		Lognormal mean	286.15
Detection limit or PQL		Std. devn.	150.9552
Method detection limit		Median	221
TOTAL	7	Min.	187
		Max.	601

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Reject lognormal distribution.
W value is 0.7885. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.7112. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 396.72

0.025   Porcupine Bay
0.025   Mercury
0.025
0.025
0.025
0.025
0.025

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.03	
Censored		Lognormal mean	0.03	
Detection limit or PQL		Std. devn.	3.8E-10	
Method detection limit		Median	0.025	
TOTAL	7	Min.	0.025	
		Max.	0.025	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.03

100	Porcupine Bay
129	Zinc
214	

		MTCA*Stat* 2.1		
129	Number of samples		Uncensored values	
113	Uncensored	7	Mean	136.71
111	Censored		Lognormal mean	137.11
161	Detection limit or PQL		Std. devn.	39.27134
	Method detection limit		Median	129
	TOTAL	7	Min.	100
			Max.	214

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.905. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 165.55

0.5
0.5
0.5
0.495
0.5
0.5
0.495

No Name Campground

Antimony

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.50	
Censored		Lognormal mean	0.50	
Detection limit or PQL		Std. devn.	0.00244	
Method detection limit		Median	0.5	
TOTAL	7	Min.	0.495	
		Max.	0.5	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.693. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.5

11.1   No Name Campground
9.2   Arsenic
11.1
10.6
9.7
8.8
8.9

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	9.91	
Censored		Lognormal mean	9.92	
Detection limit or PQL		Std. devn.	1.009007	
Method detection limit		Median	9.7	
TOTAL	7	Min.	8.8	
		Max.	11.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8674. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 10.66

0.1   No Name Campground
0.105   Cadmium
0.1
0.1
0.1
0.1
0.1

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.10	
Censored		Lognormal mean	0.10	
Detection limit or PQL		Std. devn.	0.00189	
Method detection limit		Median	0.1	
TOTAL	7	Min.	0.1	
		Max.	0.105	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.4411. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.1

22100
19000
22100
22400
21000
21700
17900

No Name Campground

Iron

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	20885.71
Censored			Lognormal mean	20898.70
Detection limit or PQL			Std. devn.	1750.646
Method detection limit			Median	21700
TOTAL	7		Min.	17900
			Max.	22400

Lognormal distribution?　　Normal distribution?
r-squared is:　　　　　　　r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8183. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 22171.36

16.9
15.8
15.9
12.8
11.7
12.8
13.1

No Name Campground

Lead

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	14.14
Censored			Lognormal mean	14.16
Detection limit or PQL			Std. devn.	2.004044
Method detection limit			Median	13.1
TOTAL	7		Min.	11.7
			Max.	16.9

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8854. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 15.61

529   No Name Campground
402   Manganese
501
493
405
484
474

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	469.71	
Censored		Lognormal mean	470.14	
Detection limit or PQL		Std. devn.	48.35878	
Method detection limit		Median	484	
TOTAL	7	Min.	402	
		Max.	529	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8587. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 505.23

0.05
0.05
0.05
0.05
0.05
0.05
0.05

No Name Campground

Mercury

MTCA*Stat* 2.1			
Number of samples		Uncensored values	
Uncensored	7	Mean	0.05
Censored		Lognormal mean	0.05
Detection limit or PQL		Std. devn.	7.6E-10
Method detection limit		Median	0.05
TOTAL	7	Min.	0.05
		Max.	0.05

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.05

120
117
91.8
94.5
98.9
76.3
84.6

No Name Campground

Zinc

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	97.59
Censored		Lognormal mean	97.77
Detection limit or PQL		Std. devn.	16.05069
Method detection limit		Median	94.5
TOTAL	7	Min.	76.3
		Max.	120

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9513. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 109.37

0.49
0.5
0.5
0.485
0.5
0.485
0.485

Horseshoe Point

Antimony

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.49	
Censored		Lognormal mean	0.49	
Detection limit or PQL		Std. devn.	0.007559	
Method detection limit		Median	0.49	
TOTAL	7	Min.	0.485	
		Max.	0.5	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.7786. This is less than the tabled value of 0.803
Assume normal distribution.
W value is 0.875. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.5

12.1
18.3
5.3
9
14.9
12
9.7

Horseshoe Point Campground

Arsenic

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	11.61	
Censored		Lognormal mean	11.81	
Detection limit or PQL		Std. devn.	4.208099	
Method detection limit		Median	12	
TOTAL	7	Min.	5.3	
		Max.	18.3	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9558. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 14.7

0.1
0.1
0.1
0.095
0.1
0.1
0.095

Horseshoe Point Campground

Cadmium

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.10	
Censored		Lognormal mean	0.10	
Detection limit or PQL		Std. devn.	0.00244	
Method detection limit		Median	0.1	
TOTAL	7	Min.	0.095	
		Max.	0.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.6119. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 0.1

17900
19500
13300
14400
19600
18900
17400

Horseshoe Point Campground

Iron

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	17285.71	
Censored		Lognormal mean	17321.39	
Detection limit or PQL		Std. devn.	2498.952	
Method detection limit		Median	17900	
TOTAL	7	Min.	13300	
		Max.	19600	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8449. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 19120.91

12.3
15.2
 7.6
10.6
13.8
12.7
11.3

Horseshoe Point Campground

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	11.93	
Censored		Lognormal mean	11.99	
Detection limit or PQL		Std. devn.	2.443846	
Method detection limit		Median	12.3	
TOTAL	7	Min.	7.6	
		Max.	15.2	

Lognormal distribution? Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9243. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 13.72

450
412
242
237
377
400
344

Horseshoe Point Campground

Manganese

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	351.71
Censored			Lognormal mean	353.94
Detection limit or PQL			Std. devn.	83.18797
Method detection limit			Median	377
TOTAL	7		Min.	237
			Max.	450

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.8509. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 412.81

0.05   Horseshoe Point Campground
0.05   Mercury
0.05
0.05

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.05	
Censored		Lognormal mean	0.05	
Detection limit or PQL		Std. devn.	7.6E-10	
Method detection limit		Median	0.05	
TOTAL	7	Min.	0.05	
		Max.	0.05	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.05

81.5
55.9
60.1
104
70.8
81.5
72.8

Horseshoe Point Campground

Zinc

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	75.23
Censored			Lognormal mean	75.44
Detection limit or PQL			Std. devn.	15.99184
Method detection limit			Median	72.8
TOTAL	7		Min.	55.9
			Max.	104

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9628. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 86.97

0.49
0.495
0.495
0.485
0.5
0.5
0.49

Pierre Campground

Antimony

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.49
Censored		Lognormal mean	0.49
Detection limit or PQL		Std. devn.	0.005563
Method detection limit		Median	0.495
TOTAL	7	Min.	0.485
		Max.	0.5

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.9163. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 0.5

5.8
6.8
5.7
8.4
6.9
7.9
12.2

Pierre Campground

Arsenic

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	7.67
Censored			Lognormal mean	7.69
Detection limit or PQL			Std. devn.	2.229884
Method detection limit			Median	6.9
TOTAL	7		Min.	5.7
			Max.	12.2

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9012. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 9.31

0.1 Pierre Campground
0.1 Cadmium
0.1
0.1
0.1
0.1
0.1

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.10	
Censored		Lognormal mean	0.10	
Detection limit or PQL		Std. devn.	1.52E-09	
Method detection limit		Median	0.1	
TOTAL	7	Min.	0.1	
		Max.	0.1	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.1

14000
12700
13200
18100
15200
18000
23300

Pierre Campground

Iron

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	16357.14
Censored			Lognormal mean	16399.89
Detection limit or PQL			Std. devn.	3745.601
Method detection limit			Median	15200
TOTAL	7		Min.	12700
			Max.	23300

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9219. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 19107.86

11.7   Pierre Campground
 8.5   Lead
 9.7
11.5
10.4
11.7
14.5

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	11.14	
Censored		Lognormal mean	11.17	
Detection limit or PQL		Std. devn.	1.898997	
Method detection limit		Median	11.5	
TOTAL	7	Min.	8.5	
		Max.	14.5	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.963. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 12.54

208   Pierre Campground
292   Manganese
164
322
299
458
660

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	343.29	
Censored		Lognormal mean	347.88	
Detection limit or PQL		Std. devn.	167.8081	
Method detection limit		Median	299	
TOTAL	7	Min.	164	
		Max.	660	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9708. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 466.52

0.05
0.05   Pierre Campground
0.05   Mercury
0.05
0.05
0.05
0.05

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.05	
Censored		Lognormal mean	0.05	
Detection limit or PQL		Std. devn.	7.6E-10	
Method detection limit		Median	0.05	
TOTAL	7	Min.	0.05	
		Max.	0.05	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.05

190   Pierre Campground
52.9   Zinc
209
202
194
95.9
79.5

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	146.19	
Censored		Lognormal mean	151.89	
Detection limit or PQL		Std. devn.	67.01322	
Method detection limit		Median	190	
TOTAL	7	Min.	52.9	
		Max.	209	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:
Assume lognormal distribution.
W value is 0.8207. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 195.4

4.2
8.5
6.6
6.4
5.4
6
4.2

Fort Spokane

Arsenic

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	5.90	
Censored		Lognormal mean	5.93	
Detection limit or PQL		Std. devn.	1.502221	
Method detection limit		Median	6	
TOTAL	7	Min.	4.2	
		Max.	8.5	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9326. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 7

0.05
0.05
0.05
0.05
0.05
0.05
0.05

Fort Spokane

Cadmium

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	0.05
Censored		Lognormal mean	0.05
Detection limit or PQL		Std. devn.	7.6E-10
Method detection limit		Median	0.05
TOTAL	7	Min.	0.05
		Max.	0.05

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.05

9080
11500
10000
11600
11400
11600
8560

Fort Spokane

Iron

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	10534.29	
Censored		Lognormal mean	10548.78	
Detection limit or PQL		Std. devn.	1307.145	
Method detection limit		Median	11400	
TOTAL	7	Min.	8560	
		Max.	11600	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Reject lognormal distribution.
W value is 0.8019. This is less than the tabled value of 0.803
Reject normal distribution.
W value is 0.8028. This is less than the tabled value of 0.803

UCL (based on t-statistic) is 11494.23

7.2
9.2
8.4
12.4
8.7
8.9
5.8

Fort Spokane

Lead

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	8.66	
Censored		Lognormal mean	8.69	
Detection limit or PQL		Std. devn.	2.029661	
Method detection limit		Median	8.7	
TOTAL	7	Min.	5.8	
		Max.	12.4	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9507. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 10.15

190   Fort Spokane
232   Manganese
238
270
247
247
202

MTCA*Stat* 2.1				
Number of samples		Uncensored values		
Uncensored	7	Mean	232.29	
Censored		Lognormal mean	232.57	
Detection limit or PQL		Std. devn.	27.66896	
Method detection limit		Median	238	
TOTAL	7	Min.	190	
		Max.	270	

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9227. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 252.61

0.025
0.025
0.025
0.025   Fort Spokane
0.025   Mercury
0.025
0.025

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	0.03
Censored			Lognormal mean	0.03
Detection limit or PQL			Std. devn.	3.8E-10
Method detection limit			Median	0.025
TOTAL	7		Min.	0.025
			Max.	0.025

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:
Recommendations:

UCL (based on t-statistic) is 0.03

47.5
39.7
34.1
100
53.2
61
26.5

Fort Spokane

Zinc

MTCA*Stat* 2.1				
Number of samples			Uncensored values	
Uncensored	7		Mean	51.71
Censored			Lognormal mean	52.21
Detection limit or PQL			Std. devn.	24.24895
Method detection limit			Median	47.5
TOTAL	7		Min.	26.5
			Max.	100

Lognormal distribution?   Normal distribution?
r-squared is: r-squared is:

Recommendations:
Assume lognormal distribution.
W value is 0.9819. This exceeds the tabled value of 0.803

UCL (based on t-statistic) is 69.52

	STATISTC$	SB201	RSB201	PCTILE
1	Mean	3.214	475.500	95.000
2	Mean	3.214	475.500	95.000
3	Mean	3.229	477.000	95.300
4	Mean	3.243	478.000	95.500
5	Mean	3.257	479.000	95.700
6	Mean	3.257	480.500	96.000
7	Mean	3.257	480.500	96.000

	STATISTC$	AS201	RAS201	PCTILE
1	Mean	29.343	475.500	95.000
2	Mean	29.343	475.500	95.000
3	Mean	29.543	477.000	95.300
4	Mean	29.557	478.000	95.500
5	Mean	29.571	479.000	95.700
6	Mean	29.586	480.500	96.000
7	Mean	29.586	480.500	96.000

	STATISTC$	AS201	RAS201	PCTILE
1	Mean	50.986	475.500	95.000
2	Mean	50.986	475.500	95.000
3	Mean	51.000	477.000	95.300
4	Mean	51.014	478.000	95.500
5	Mean	51.057	479.000	95.700
6	Mean	51.471	480.500	96.000
7	Mean	51.471	480.500	96.000

	STATISTC$	CD201	RCD201	PCTILE
1	Mean	17.600	475.500	95.000
2	Mean	17.600	475.500	95.000
3	Mean	17.614	477.000	95.300
4	Mean	17.657	478.000	95.500
5	Mean	17.671	479.000	95.700
6	Mean	17.686	480.500	96.000
7	Mean	17.686	480.500	96.000

	STATISTC$	FE201	RFE201	PCTILE
1	Mean	27285.714	477.500	95.400
2	Mean	27285.714	477.500	95.400
3	Mean	27285.714	477.500	95.400
4	Mean	27285.714	477.500	95.400

	STATISTC$	MN201	RMN201	PCTILE
1	Mean	2548.571	476.500	95.200
2	Mean	2548.571	476.500	95.200
3	Mean	2550.000	478.000	95.500
4	Mean	2554.286	479.000	95.700
5	Mean	2555.714	480.000	95.900

	STATISTC$	HG201	RHG201	PCTILE
1	Mean	0.380	477.000	95.300
2	Mean	0.380	477.000	95.300
3	Mean	0.380	477.000	95.300
4	Mean	0.381	479.000	95.700

	STATISTC$	ZN201	RZN201	PCTILE
1	Mean	3022.857	476.500	95.200
2	Mean	3022.857	476.500	95.200
3	Mean	3032.857	478.000	95.500
4	Mean	3035.714	479.000	95.700
5	Mean	3040.000	480.000	95.900

	STATISTC$	SB202	RSB202	PCTILE
1	Mean	2.086	475.500	95.000
2	Mean	2.086	475.500	95.000
3	Mean	2.100	477.000	95.300
4	Mean	2.114	479.000	95.700
5	Mean	2.114	479.000	95.700
6	Mean	2.114	479.000	95.700

	STATISTC$	AS202	RAS202	PCTILE
1	Mean	20.157	476.500	95.200
2	Mean	20.157	476.500	95.200
3	Mean	20.229	478.000	95.500
4	Mean	20.271	480.000	95.900
5	Mean	20.271	480.000	95.900
6	Mean	20.271	480.000	95.900

	STATISTC$	CD202	RCD202	PCTILE
1	Mean	10.586	476.000	95.100
2	Mean	10.614	477.000	95.300
3	Mean	10.671	480.000	95.900
4	Mean	10.671	480.000	95.900
5	Mean	10.671	480.000	95.900
6	Mean	10.671	480.000	95.900
7	Mean	10.671	480.000	95.900

	STATISTC$	FE202	RFE202	PCTILE
1	Mean	28942.857	476.500	95.200
2	Mean	28942.857	476.500	95.200
3	Mean	28957.143	478.000	95.500
4	Mean	28971.429	480.000	95.900
5	Mean	28971.429	480.000	95.900
6	Mean	28971.429	480.000	95.900

	STATISTC$	MN202	RMN202	PCTILE
1	Mean	1570.000	477.000	95.300
2	Mean	1577.143	478.000	95.500
3	Mean	1580.571	479.000	95.700
4	Mean	1586.286	480.500	96.000
5	Mean	1586.286	480.500	96.000

	STATISTC$	HG202	RHG202	PCTILE
1	Mean	0.240	478.500	95.600
2	Mean	0.240	478.500	95.600
3	Mean	0.240	478.500	95.600
4	Mean	0.240	478.500	95.600
5	Mean	0.240	478.500	95.600
6	Mean	0.240	478.500	95.600

	STATISTC$	ZN202	RZN202	PCTILE
1	Mean	2240.000	477.000	95.300
2	Mean	2242.857	478.500	95.600
3	Mean	2242.857	478.500	95.600
4	Mean	2247.143	480.000	95.900

	STATISTC$	SB203	RSB203	PCTILE
1	Mean	1.557	476.500	95.200
2	Mean	1.557	476.500	95.200
3	Mean	1.592	478.000	95.500
4	Mean	1.620	480.500	96.000
5	Mean	1.620	480.500	96.000
6	Mean	1.620	480.500	96.000
7	Mean	1.620	480.500	96.000

	STATISTC$	AS203	RAS203	PCTILE
1	Mean	15.075	476.000	95.100
2	Mean	15.100	477.000	95.300
3	Mean	15.180	478.500	95.600
4	Mean	15.180	478.500	95.600
5	Mean	15.200	480.500	96.000
6	Mean	15.200	480.500	96.000

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	STATISTC$	CD203	RCD203	PCTILE
1	Mean	7.457	475.500	95.000
2	Mean	7.457	475.500	95.000
3	Mean	7.500	477.000	95.300
4	Mean	7.600	478.000	95.500
5	Mean	7.614	479.000	95.700
6	Mean	7.629	480.500	96.000
7	Mean	7.629	480.500	96.000

	STATISTC$	FE203	RFE203	PCTILE
1	Mean	22800.000	475.500	95.000
2	Mean	22800.000	475.500	95.000
3	Mean	22814.286	477.500	95.400
4	Mean	22814.286	477.500	95.400
5	Mean	22842.857	479.000	95.700
6	Mean	22857.143	480.000	95.900

	STATISTC$	MN203	RMN203	PCTILE
1	Mean	1608.286	476.500	95.200
2	Mean	1608.286	476.500	95.200
3	Mean	1738.286	478.000	95.500
4	Mean	1752.000	479.000	95.700
5	Mean	1752.429	480.000	95.900

	STATISTC$	HG203	RHG203	PCTILE
1	Mean	0.117	476.500	95.200
2	Mean	0.117	476.500	95.200
3	Mean	0.122	478.500	95.600
4	Mean	0.122	478.500	95.600
5	Mean	0.122	480.000	95.900

	STATISTC$	ZN203	RZN203	PCTILE
1	Mean	2020.000	476.500	95.200
2	Mean	2020.000	476.500	95.200
3	Mean	2022.857	479.500	95.800
4	Mean	2022.857	479.500	95.800
5	Mean	2022.857	479.500	95.800
6	Mean	2022.857	479.500	95.800

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	STATISTC$	SB204	RSB204	PCTILE
1	Mean	2.686	475.500	95.000
2	Mean	2.686	475.500	95.000
3	Mean	2.686	477.500	95.400
4	Mean	2.686	477.500	95.400
5	Mean	2.700	480.500	96.000
6	Mean	2.700	480.500	96.000
7	Mean	2.700	480.500	96.000
8	Mean	2.700	480.500	96.000

	STATISTC$	AS204	RAS204	PCTILE
1	Mean	36.171	477.000	95.300
2	Mean	36.171	477.000	95.300
3	Mean	36.171	477.000	95.300
4	Mean	36.271	479.000	95.700
5	Mean	36.414	480.000	95.900

	STATISTC$	AS204	RAS204	PCTILE
1	Mean	22.986	476.000	95.100
2	Mean	23.057	477.500	95.400
3	Mean	23.057	477.500	95.400
4	Mean	23.229	479.500	95.800
5	Mean	23.229	479.500	95.800

	STATISTC$	CD204	RCD204	PCTILE
1	Mean	13.086	476.000	95.100
2	Mean	13.114	477.000	95.300
3	Mean	13.114	478.000	95.500
4	Mean	13.157	479.000	95.700
5	Mean	13.186	480.000	95.900

	STATISTC$	FE204	RFE204	PCTILE
1	Mean	40571.429	476.500	95.200
2	Mean	40571.429	476.500	95.200
3	Mean	40585.714	478.500	95.600
4	Mean	40585.714	478.500	95.600
5	Mean	40714.286	480.000	95.900

	STATISTC$	MN204	RMN204	PCTILE
1	Mean	1551.429	476.000	95.100
2	Mean	1552.714	477.000	95.300
3	Mean	1552.857	478.000	95.500
4	Mean	1555.571	479.000	95.700
5	Mean	1560.000	480.000	95.900

	STATISTC$	HG204	RHG204	PCTILE
1	Mean	0.277	476.000	95.100
2	Mean	0.281	477.000	95.300
3	Mean	0.283	478.500	95.600
4	Mean	0.283	478.500	95.600
5	Mean	0.283	480.000	95.900

	STATISTC$	ZN204	RZN204	PCTILE
1	Mean	3412.857	476.500	95.200
2	Mean	3412.857	476.500	95.200
3	Mean	3415.714	478.000	95.500
4	Mean	3438.571	479.000	95.700
5	Mean	3445.714	480.000	95.900

	STATISTC$	SB205	RSB205	PCTILE
1	Mean	1.504	480.500	96.000
2	Mean	1.504	480.500	96.000
3	Mean	1.504	480.500	96.000
4	Mean	1.504	480.500	96.000

	STATISTC$	AS205	RAS205	PCTILE
1	Mean	21.357	476.000	95.100
2	Mean	21.357	476.000	95.100
3	Mean	21.357	476.000	95.100
4	Mean	21.371	478.000	95.500
5	Mean	21.443	479.000	95.700
6	Mean	21.457	480.000	95.900

	STATISTC$	CD205	RCD205	PCTILE
1	Mean	8.743	476.000	95.100
2	Mean	8.800	477.000	95.300
3	Mean	8.829	478.500	95.600
4	Mean	8.829	478.500	95.600
5	Mean	8.843	480.000	95.900

	STATISTC$	FE205	RFE205	PCTILE
1	Mean	27357.143	476.000	95.100
2	Mean	27371.429	477.000	95.300
3	Mean	27385.714	478.000	95.500
4	Mean	27400.000	480.000	95.900
5	Mean	27400.000	480.000	95.900
6	Mean	27400.000	480.000	95.900

	STATISTC$	MN205	RMN205	PCTILE
1	Mean	1728.571	476.000	95.100
2	Mean	1732.857	477.000	95.300
3	Mean	1734.286	478.500	95.600
4	Mean	1734.286	478.500	95.600
5	Mean	1737.143	480.000	95.900

	STATISTC$	HG205	RHG205	PCTILE
1	Mean	0.136	476.500	95.200
2	Mean	0.136	476.500	95.200

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	STATISTC$	ZN205	RZN205	PCTILE
1	Mean	3808.571	477.000	95.300
2	Mean	3808.571	477.000	95.300
3	Mean	3808.571	477.000	95.300
4	Mean	3814.286	479.500	95.800
5	Mean	3814.286	479.500	95.800

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	STATISTC$	SB206	RSB206	PCTILE
1	Mean	1.021	475.500	95.000
2	Mean	1.021	475.500	95.000
3	Mean	1.021	475.500	95.000
4	Mean	1.021	475.500	95.000
5	Mean	1.029	478.500	95.600
6	Mean	1.029	478.500	95.600

	STATISTC$	AS206	RAS206	PCTILE
1	Mean	14.486	476.000	95.100
2	Mean	14.500	477.500	95.400
3	Mean	14.500	477.500	95.400
4	Mean	14.514	479.000	95.700
5	Mean	14.529	480.000	95.900

	STATISTC$	AS206	RAS206	PCTILE
1	Mean	7.171	476.500	95.200
2	Mean	7.171	476.500	95.200
3	Mean	7.171	478.000	95.500
4	Mean	7.200	479.000	95.700
5	Mean	7.214	480.000	95.900

	STATISTC$	CD206	RCD206	PCTILE
1	Mean	1.614	477.000	95.300
2	Mean	1.629	478.000	95.500
3	Mean	1.629	479.500	95.800
4	Mean	1.629	479.500	95.800

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	STATISTC$	FE206	RFE206	PCTILE
1	Mean	31028.571	477.500	95.400
2	Mean	31028.571	477.500	95.400
3	Mean	31028.571	477.500	95.400
4	Mean	31028.571	477.500	95.400
5	Mean	31057.143	480.000	95.900

	STATISTC$	MN206	RMN206	PCTILE
1	Mean	589.857	477.000	95.300
2	Mean	589.857	477.000	95.300
3	Mean	589.857	477.000	95.300
4	Mean	590.429	479.000	95.700
5	Mean	593.857	480.500	96.000
6	Mean	593.857	480.500	96.000

	STATISTC$	ZN206	RZN206	PCTILE
1	Mean	453.143	476.500	95.200
2	Mean	453.143	476.500	95.200
3	Mean	454.571	478.000	95.500
4	Mean	455.143	479.000	95.700
5	Mean	464.143	480.500	96.000
6	Mean	464.143	480.500	96.000

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	STATISTC$	AS208	RAS208	PCTILE
1	Mean	6.867	477.000	95.300
2	Mean	6.940	479.000	95.700
3	Mean	6.940	479.000	95.700
4	Mean	6.940	479.000	95.700

	STATISTC$	FE208	RFE208	PCTILE
1	Mean	18271.429	476.500	95.200
2	Mean	18271.429	476.500	95.200
3	Mean	18271.429	476.500	95.200
4	Mean	18271.429	476.500	95.200
5	Mean	18342.857	479.500	95.800
6	Mean	18342.857	479.500	95.800

	STATISTC$	MN208	RMN208	PCTILE
1	Mean	516.714	476.000	95.100
2	Mean	517.143	477.000	95.300
3	Mean	517.429	478.000	95.500
4	Mean	518.143	479.000	95.700
5	Mean	518.429	480.500	96.000
6	Mean	518.429	480.500	96.000

	STATISTC$	ZN208	RZN208	PCTILE
1	Mean	110.257	476.000	95.100
2	Mean	110.386	477.500	95.400
3	Mean	110.386	477.500	95.400
4	Mean	110.414	479.000	95.700
5	Mean	110.700	480.500	96.000
6	Mean	110.700	480.500	96.000

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	STATISTC$	SB209	RSB209	PCTILE
1	Mean	0.495	477.000	95.300
2	Mean	0.495	477.000	95.300
3	Mean	0.495	477.000	95.300
4	Mean	0.495	477.000	95.300
5	Mean	0.495	477.000	95.300
6	Mean	0.495	477.000	95.300
7	Mean	0.495	477.000	95.300
8	Mean	0.495	477.000	95.300
9	Mean	0.495	477.000	95.300
10	Mean	0.495	477.000	95.300
11	Mean	0.495	477.000	95.300
12	Mean	0.495	477.000	95.300
13	Mean	0.495	477.000	95.300
14	Mean	0.495	477.000	95.300
15	Mean	0.495	477.000	95.300
16	Mean	0.495	477.000	95.300
17	Mean	0.495	477.000	95.300

	STATISTC$	AS209	RAS209	PCTILE
1	Mean	16.014	476.000	95.100
2	Mean	16.014	476.000	95.100
3	Mean	16.014	476.000	95.100
4	Mean	16.014	478.000	95.500
5	Mean	16.029	480.500	96.000
6	Mean	16.029	480.500	96.000
7	Mean	16.029	480.500	96.000
8	Mean	16.029	480.500	96.000

	STATISTC$	FE209	RFE209	PCTILE
1	Mean	25057.143	477.000	95.300
2	Mean	25057.143	477.000	95.300
3	Mean	25057.143	477.000	95.300
4	Mean	25100.000	479.000	95.700

	STATISTC$	MN209	RMN209	PCTILE
1	Mean	437.714	476.000	95.100
2	Mean	438.286	477.000	95.300
3	Mean	438.714	478.000	95.500
4	Mean	440.000	479.000	95.700
5	Mean	441.143	480.000	95.900

	STATISTC$	ZN209	RZN209	PCTILE
1	Mean	100.400	476.000	95.100
2	Mean	101.243	477.000	95.300
3	Mean	101.657	478.000	95.500
4	Mean	101.714	479.000	95.700
5	Mean	102.371	480.000	95.900

	STATISTC$	SB210	RSB210	PCTILE
1	Mean	0.871	479.500	95.800
2	Mean	0.871	479.500	95.800
3	Mean	0.871	479.500	95.800
4	Mean	0.871	479.500	95.800
5	Mean	0.871	479.500	95.800
6	Mean	0.871	479.500	95.800

	STATISTC$	AS210	RAS210	PCTILE
1	Mean	11.750	475.500	95.000
2	Mean	11.750	475.500	95.000
3	Mean	11.750	475.500	95.000
4	Mean	11.750	475.500	95.000
5	Mean	11.800	479.000	95.700
6	Mean	11.800	479.000	95.700
7	Mean	11.800	479.000	95.700

	STATISTC$	AS210	RAS210	PCTILE
1	Mean	10.514	476.500	95.200
2	Mean	10.514	476.500	95.200
3	Mean	10.514	476.500	95.200
4	Mean	10.514	476.500	95.200
5	Mean	10.543	479.000	95.700
6	Mean	10.600	480.000	95.900

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	STATISTC$	CD210	RCD210	PCTILE
1	Mean	1.789	476.000	95.100
2	Mean	1.794	477.000	95.300
3	Mean	1.796	478.000	95.500
4	Mean	1.803	479.000	95.700
5	Mean	1.806	480.000	95.900

	STATISTC$	FE210	RFE210	PCTILE
1	Mean	15457.143	477.000	95.300
2	Mean	15457.143	477.000	95.300
3	Mean	15457.143	477.000	95.300
4	Mean	15485.714	479.500	95.800
5	Mean	15485.714	479.500	95.800

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	STATISTC$	MN210	RMN210	PCTILE
1	Mean	241.857	476.000	95.100
2	Mean	241.857	476.000	95.100
3	Mean	241.857	476.000	95.100
4	Mean	242.286	478.000	95.500
5	Mean	242.429	479.000	95.700
6	Mean	243.429	480.000	95.900

	STATISTC$	HG210	RHG210	PCTILE
1	Mean	0.207	476.500	95.200
2	Mean	0.207	476.500	95.200
3	Mean	0.230	478.000	95.500
4	Mean	0.233	479.000	95.700
5	Mean	0.236	480.500	96.000
6	Mean	0.236	480.500	96.000

	STATISTC$	ZN210	RZN210	PCTILE
1	Mean	354.571	476.000	95.100
2	Mean	354.857	477.000	95.300
3	Mean	355.000	478.000	95.500
4	Mean	357.143	479.000	95.700
5	Mean	357.429	480.500	96.000
6	Mean	357.429	480.500	96.000

	STATISTC$	SB217	RSB217	PCTILE
1	Mean	0.512	476.000	95.100
2	Mean	0.513	477.500	95.400
3	Mean	0.513	477.500	95.400
4	Mean	0.514	479.500	95.800
5	Mean	0.514	479.500	95.800

	STATISTC$	AS217	RAS217	PCTILE
1	Mean	10.443	476.000	95.100
2	Mean	10.443	477.000	95.300
3	Mean	10.486	478.000	95.500
4	Mean	10.500	480.000	95.900
5	Mean	10.500	480.000	95.900
6	Mean	10.500	480.000	95.900

	STATISTC$	FE217	RFE217	PCTILE
1	Mean	20828.571	477.000	95.300
2	Mean	20828.571	477.000	95.300
3	Mean	20828.571	477.000	95.300
4	Mean	20842.857	479.500	95.800
5	Mean	20842.857	479.500	95.800

	STATISTC$	MN217	RMN217	PCTILE
1	Mean	476.714	476.000	95.100
2	Mean	476.857	478.000	95.500
3	Mean	476.857	478.000	95.500
4	Mean	476.857	478.000	95.500
5	Mean	477.429	480.000	95.900

	STATISTC$	HG217	RHG217	PCTILE
1	Mean	0.173	476.500	95.200
2	Mean	0.173	476.500	95.200
3	Mean	0.173	476.500	95.200
4	Mean	0.173	476.500	95.200
5	Mean	0.173	476.500	95.200
6	Mean	0.173	476.500	95.200
7	Mean	0.179	480.000	95.900

	STATISTC$	ZN217	RZN217	PCTILE
1	Mean	117.200	475.500	95.000
2	Mean	117.200	475.500	95.000
3	Mean	117.300	477.000	95.300
4	Mean	117.357	478.000	95.500
5	Mean	117.457	479.000	95.700
6	Mean	117.557	480.000	95.900

	STATISTC$	AS218	RAS218	PCTILE
1	Mean	9.917	476.500	95.200
2	Mean	9.917	476.500	95.200
3	Mean	9.920	478.000	95.500
4	Mean	9.925	479.500	95.800
5	Mean	9.925	479.500	95.800

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	STATISTC$	AS218	RAS218	PCTILE
1	Mean	5.614	478.500	95.600
2	Mean	5.614	478.500	95.600
3	Mean	5.629	480.000	95.900

	STATISTC$	FE218	RFE218	PCTILE
1	Mean	19342.857	476.000	95.100
2	Mean	19357.143	477.000	95.300
3	Mean	19371.429	479.000	95.700
4	Mean	19371.429	479.000	95.700
5	Mean	19371.429	479.000	95.700

	STATISTC$	MN218	RMN218	PCTILE
1	Mean	297.000	477.500	95.400
2	Mean	297.000	477.500	95.400
3	Mean	297.000	477.500	95.400
4	Mean	297.000	477.500	95.400
5	Mean	297.000	477.500	95.400
6	Mean	297.000	477.500	95.400

	STATISTC$	ZN218	RZN218	PCTILE
1	Mean	225.714	476.000	95.100
2	Mean	227.000	477.500	95.400
3	Mean	227.000	477.500	95.400
4	Mean	227.714	479.000	95.700
5	Mean	228.714	480.000	95.900

	STATISTC$	AS219	RAS219	PCTILE
1	Mean	9.686	476.500	95.200
2	Mean	9.686	476.500	95.200
3	Mean	9.700	478.000	95.500
4	Mean	9.757	479.000	95.700
5	Mean	9.771	480.000	95.900

	STATISTC$	CD219	RCD219	PCTILE
1	Mean	0.104	476.000	95.100
2	Mean	0.104	476.000	95.100
3	Mean	0.104	476.000	95.100
4	Mean	0.131	478.000	95.500

	STATISTC$	FE219	RFE219	PCTILE
1	Mean	25742.857	477.000	95.300
2	Mean	25742.857	477.000	95.300
3	Mean	25742.857	477.000	95.300
4	Mean	25757.143	479.500	95.800
5	Mean	25757.143	479.500	95.800

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	STATISTC$	MN219	RMN219	PCTILE
1	Mean	367.571	476.000	95.100
2	Mean	367.571	476.000	95.100
3	Mean	367.571	476.000	95.100
4	Mean	369.429	478.500	95.600
5	Mean	369.429	478.500	95.600
6	Mean	370.000	480.500	96.000
7	Mean	370.000	480.500	96.000

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	STATISTC$	ZN219	RZN219	PCTILE
1	Mean	158.814	477.000	95.300
2	Mean	158.814	477.000	95.300
3	Mean	158.814	477.000	95.300
4	Mean	158.886	479.000	95.700
5	Mean	159.257	480.000	95.900

	STATISTC$	AS220	RAS220	PCTILE
1	Mean	15.557	476.000	95.100
2	Mean	15.586	477.000	95.300
3	Mean	16.043	478.000	95.500
4	Mean	16.114	479.500	95.800
5	Mean	16.114	479.500	95.800

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	STATISTC$	FE220	RFE220	PCTILE
1	Mean	25800.000	475.500	95.000
2	Mean	25800.000	475.500	95.000
3	Mean	25814.286	478.500	95.600
4	Mean	25814.286	478.500	95.600
5	Mean	25814.286	478.500	95.600
6	Mean	25814.286	478.500	95.600

	STATISTC$	MN220	RMN220	PCTILE
1	Mean	480.286	476.000	95.100
2	Mean	481.143	477.000	95.300
3	Mean	481.714	478.000	95.500
4	Mean	482.000	479.000	95.700
5	Mean	482.714	480.000	95.900

	STATISTC$	ZN220	RZN220	PCTILE
1	Mean	142.200	476.000	95.100
2	Mean	142.643	477.000	95.300
3	Mean	143.029	478.000	95.500
4	Mean	143.286	479.000	95.700
5	Mean	143.343	480.000	95.900

	STATISTC$	AS221	RAS221	PCTILE
1	Mean	10.857	476.000	95.100
2	Mean	10.883	477.000	95.300
3	Mean	10.900	478.000	95.500
4	Mean	10.967	479.000	95.700
5	Mean	10.971	480.000	95.900

	STATISTC$	AS221	RAS221	PCTILE
1	Mean	9.400	475.500	95.000
2	Mean	9.400	475.500	95.000
3	Mean	9.414	477.000	95.300
4	Mean	9.429	478.500	95.600
5	Mean	9.429	478.500	95.600
6	Mean	9.443	480.500	96.000
7	Mean	9.443	480.500	96.000

	STATISTC$	FE221	RFE221	PCTILE
1	Mean	16571.429	477.500	95.400
2	Mean	16571.429	477.500	95.400
3	Mean	16571.429	477.500	95.400
4	Mean	16571.429	477.500	95.400

	STATISTC$	MN221	RMN221	PCTILE
1	Mean	367.429	476.500	95.200
2	Mean	367.429	476.500	95.200
3	Mean	368.857	478.000	95.500
4	Mean	370.571	479.000	95.700
5	Mean	374.429	480.000	95.900

	STATISTC$	ZN221	RZN221	PCTILE
1	Mean	162.143	476.000	95.100
2	Mean	162.429	477.500	95.400
3	Mean	162.429	477.500	95.400
4	Mean	162.857	480.000	95.900
5	Mean	162.857	480.000	95.900
6	Mean	162.857	480.000	95.900

	STATISTC$	AS222	RAS222	PCTILE
1	Mean	10.500	477.500	95.400
2	Mean	10.500	477.500	95.400
3	Mean	10.500	477.500	95.400
4	Mean	10.500	477.500	95.400
5	Mean	10.514	480.500	96.000
6	Mean	10.514	480.500	96.000

	STATISTC$	FE222	RFE222	PCTILE
1	Mean	21914.286	478.000	95.500
2	Mean	21914.286	478.000	95.500
3	Mean	21914.286	478.000	95.500
4	Mean	21914.286	478.000	95.500
5	Mean	21914.286	478.000	95.500

	STATISTC$	MN222	RMN222	PCTILE
1	Mean	494.286	476.000	95.100
2	Mean	494.857	477.000	95.300
3	Mean	495.000	478.000	95.500
4	Mean	495.286	479.000	95.700
5	Mean	495.286	480.000	95.900

	STATISTC$	ZN222	RZN222	PCTILE
1	Mean	105.871	476.000	95.100
2	Mean	106.186	477.000	95.300
3	Mean	106.243	478.000	95.500
4	Mean	106.300	479.000	95.700
5	Mean	106.414	480.500	96.000
6	Mean	106.414	480.500	96.000

	STATISTC$	AS223	RAS223	PCTILE
1	Mean	13.886	477.500	95.400
2	Mean	13.886	477.500	95.400
3	Mean	13.900	479.000	95.700
4	Mean	14.029	480.000	95.900

	STATISTC$	CD223	RCD223	PCTILE
1	Mean	0.100	480.500	96.000
2	Mean	0.100	480.500	96.000
3	Mean	0.100	480.500	96.000
4	Mean	0.100	480.500	96.000
5	Mean	0.100	480.500	96.000
6	Mean	0.100	480.500	96.000
7	Mean	0.100	480.500	96.000
8	Mean	0.100	480.500	96.000
9	Mean	0.100	480.500	96.000
10	Mean	0.100	480.500	96.000
11	Mean	0.100	480.500	96.000
12	Mean	0.100	480.500	96.000
13	Mean	0.100	480.500	96.000
14	Mean	0.100	480.500	96.000
15	Mean	0.100	480.500	96.000
16	Mean	0.100	480.500	96.000
17	Mean	0.100	480.500	96.000
18	Mean	0.100	480.500	96.000
19	Mean	0.100	480.500	96.000
20	Mean	0.100	480.500	96.000
21	Mean	0.100	480.500	96.000
22	Mean	0.100	480.500	96.000
23	Mean	0.100	480.500	96.000
24	Mean	0.100	480.500	96.000
25	Mean	0.100	480.500	96.000
26	Mean	0.100	480.500	96.000
27	Mean	0.100	480.500	96.000
28	Mean	0.100	480.500	96.000
29	Mean	0.100	480.500	96.000
30	Mean	0.100	480.500	96.000

	STATISTC$	FE223	RFE223	PCTILE
1	Mean	18614.286	477.000	95.300
2	Mean	18614.286	477.000	95.300
3	Mean	18614.286	477.000	95.300
4	Mean	18642.857	479.000	95.700
5	Mean	18685.714	480.500	96.000
6	Mean	18685.714	480.500	96.000

	STATISTC$	MN223	RMN223	PCTILE
1	Mean	393.571	476.000	95.100
2	Mean	394.286	477.000	95.300
3	Mean	394.571	478.000	95.500
4	Mean	394.857	479.000	95.700

	STATISTC$	ZN223	RZN223	PCTILE
1	Mean	83.629	476.000	95.100
2	Mean	83.629	476.000	95.100
3	Mean	83.629	476.000	95.100
4	Mean	83.629	476.000	95.100
5	Mean	83.629	476.000	95.100
6	Mean	83.629	476.000	95.100
7	Mean	83.629	476.000	95.100
8	Mean	83.829	480.000	95.900

	STATISTC$	AS224	RAS224	PCTILE
1	Mean	8.957	476.500	95.200
2	Mean	8.957	476.500	95.200
3	Mean	8.971	478.500	95.600
4	Mean	8.971	478.500	95.600
5	Mean	9.000	480.000	95.900

	STATISTC$	FE224	RFE224	PCTILE
1	Mean	18400.000	476.000	95.100
2	Mean	18442.857	477.000	95.300
3	Mean	18457.143	478.000	95.500
4	Mean	18500.000	479.000	95.700
5	Mean	18557.143	480.500	96.000
6	Mean	18557.143	480.500	96.000

	STATISTC$	MN224	RMN224	PCTILE
1	Mean	434.857	475.500	95.000
2	Mean	434.857	475.500	95.000
3	Mean	437.857	477.000	95.300
4	Mean	441.143	478.000	95.500
5	Mean	441.143	479.000	95.700
6	Mean	443.000	480.500	96.000
7	Mean	443.000	480.500	96.000

	STATISTC$	ZN224	RZN224	PCTILE
1	Mean	183.071	475.500	95.000
2	Mean	183.071	475.500	95.000
3	Mean	183.271	477.500	95.400
4	Mean	183.271	477.500	95.400
5	Mean	183.500	479.000	95.700
6	Mean	183.700	480.000	95.900

	STATISTC$	AS225	RAS225	PCTILE
1	Mean	6.740	476.000	95.100
2	Mean	6.757	477.000	95.300
3	Mean	6.775	478.000	95.500
4	Mean	6.780	479.000	95.700
5	Mean	6.825	480.000	95.900

	STATISTC$	AS225	RAS225	PCTILE
1	Mean	5.657	476.000	95.100
2	Mean	5.657	476.000	95.100
3	Mean	5.657	476.000	95.100
4	Mean	5.671	479.000	95.700
5	Mean	5.671	479.000	95.700
6	Mean	5.671	479.000	95.700

	STATISTC$	FE225	RFE225	PCTILE
1	Mean	11182.857	477.000	95.300
2	Mean	11182.857	477.000	95.300
3	Mean	11182.857	477.000	95.300
4	Mean	11182.857	477.000	95.300
5	Mean	11182.857	477.000	95.300
6	Mean	11197.143	480.500	96.000
7	Mean	11197.143	480.500	96.000

	STATISTC$	MN225	RMN225	PCTILE
1	Mean	246.857	476.500	95.200
2	Mean	246.857	476.500	95.200
3	Mean	246.857	478.000	95.500
4	Mean	247.000	479.500	95.800
5	Mean	247.000	479.500	95.800

	STATISTC$	ZN225	RZN225	PCTILE
1	Mean	65.900	477.500	95.400
2	Mean	65.900	477.500	95.400
3	Mean	66.057	479.500	95.800
4	Mean	66.057	479.500	95.800

APPENDIX E

**Summary Intake Factors and RBC Calculations
for Chemicals Other Than Lead**

DRAFT FINAL SCREENING LEVEL HHRA
SPOKANE RIVER, WASHINGTON
Coeur d'Alene Basin RI/FS
RAC, EPA Region 10
Work Assignment No. 027-RJ-CO-102Q

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**APPENDIX E
Summary Intake Factors and RBC Calculations
for Chemicals Other Than Lead**

RBCs that are protective of human health were calculated for each metal of concern except lead.
RBCs are calculated by defining a target risk goal, then solving basic cancer and noncancer
risk assessment equations for soil concentration rather than for risk. The spreadsheet contains the
exposure assumptions, the summary intake factors, the EPA toxicity criteria, and the equations
used in the calculations. In addition, the spreadsheet contains the Spokane Area background
concentrations for each of the metals of concern.

Appendix E
Risk Based Concentrations

Chemical	Spokane RBCs Ingestion, HQ of 0.1 mg/kg	Region 9 PRGs Residential Exposure: Ingestion, Inhalation, and Dermal HQ of 1.0; mg/kg	Metals Concentrations (Ecology, 1994) Spokane Area Background
Antimony	23	31	none available
Arsenic $_{cancer}$ [b]	10 [c]	0.4	10
Arsenic $_{noncancer}$ [b]	15	22	--
Cadmium[b]	49	37	0.7
Lead	700 [e]	400	16
Iron	27,000 [d]	23,000	27,000
Manganese	7,984	1,800	769
Mercury	17	23	0.1
Zinc	17,109	23,000	71

NOTES

Target arsenic cancer risk goal of 1 x 10-6 for Region 9 PRGs and Washington State.

Arsenic RBCs were calculated assuming 100% gastrointestinal absorption.

a Assumes soil lead concentration at home is 200 ppm.

b Arsenic and cadmium include the dermal pathway.

c Arsenic's calculated RBC based on cancer risks and a 1 x 10-6 risk goal is 3 mg/kg which is below background; therefore,

   the RBC becomes 10 mg/kg, the background value of arsenic.

d Iron's calculated RBC is below background; therefore, the RBC becomes 27,000 mg/kg, the background value of iron for the area.

e Based on 200 ppm residential soil lead concentrations and a 2/3 weighting of soil ingestion at the beach.

Washington State Department of Ecology (Ecology), 1994. Natural Background Soil Metals Concentrations in Washington State.
  Toxics Cleanup Program, #94-115. October.

Appendix E
Risk Based Concentrations

Summary Intake Factors

For Chemicals Other Than Lead

Exposure Pathway	Contact Rate a	Conversion Factor CF soil - kg/mg;	Event Time EV hr/event	Event Frequency EF days/year	Exposure Duration ED years	Body Weight BW kg	Averaging Time - AT days		Summary Intake Factor SIF	
							noncarcinogens ATn	carcinogens ATc	noncarcinogens	carcinogens
Soil Ingestion	300 mg/day, child	1.0E-06	--	32	6	15	2,190	25,550	1.75E-06	1.93E-07
	100 mg/day, adult			32	24	70				
Dermal Contact - Soil	6,500 cm2, child	1.0E-06	--	32	6	15	2,190	25,550	3.80E-05	1.42E-06
	18,000 cm2, adult			32	24	70				

NOTES:

-- Not applicable

a) Contact rate is either ingestion rate for soil (IRS); surface area (SA) for dermal.

Lower case "a" and "c" notations in the formulas below indicate adult and child values, respectively. Rows are identified as containing child or adult values

in the column after the contact rate value.

SOIL SIFs:
      Soil Ingestion SIF, child cancer = CF x EFc x [(EDc x IRSc / BWc) + (EDa x IRSa / BWa)] / ATc

      Soil Ingestion SIF, child non-cancer = CF x [(EFc x EDc x IRSc) / BWc] / ATn

      Dermal Contact SIFsoil, child cancer = CF x EFc x [(EDc x SAc / BWc) + (EDa x SAa / BWa)] / ATc

      Dermal Contact SIFsoil, child non-cancer = CF x [(EFc x EDc x SAc) / BWc] / ATn

RBC Calculations

For Chemicals Other Than Lead

Chemical	Dermal Absorption ABSd	Adherence Factor (AF) Children	Adherence Factor (AF) Adult	Reference Dose RfD	Hazard Quotient HQ	Target Risk	Slope Factor SF
		(mg/cm2)	(mg/cm2)	(mg/kg/day)	unitless	unitless	(mg/kg/day)-1
Antimony	0.00	0.2	0.1	0.0004	0.1	n/a	--
Arsenic $_{cancer}$	0.03	0.2	0.1	--	n/a	0.000001	1.5
Arsenic $_{noncancer}$	0.03	0.2	0.1	0.0003	0.1	n/a	--
Cadmium$_{food-oral}$	0.00	0.2	0.1	0.001	0.1	n/a	--
Cadmium$_{food-dermal}$	0.001	0.2	0.1	0.000025	0.1	n/a	--
Iron	0.00	0.2	0.1	0.3	0.1	n/a	--
Manganese	0.00	0.2	0.1	0.14	0.1	n/a	--
Mercury	0.00	0.2	0.1	0.0003	0.1	n/a	--
Zinc	0.00	0.2	0.1	0.3	0.1	n/a	--

Chemical	Spokane River RBCs Ingestion and Dermal (mg/kg)
Antimony	22.8
Arsenic $_{cancer}$	2.8
Arsenic $_{noncancer}$	15.1
Cadmium $_{food}$ *	48.6
Iron	17,109.4
Manganese	7,984.4
Mercury	17.1
Zinc	17,109.4

NOTES: -- not available;   n/a: Not Applicable

Combined Soil Ingestion & Dermal RBCs, cancer endpoint = Target Risk / SF x [(SIF soil ing x ABS)+(SIF soil dermal x AF x ABSd)]

Combined Soil Ingestion & Dermal Exposure RBCs, non-cancer endpoint = Target Hazard x RfD / [(SIF soil ing x ABS)+(SIF soil dermal x AF x ABSd)]

ABS = gastrointestinal absorption, for all chemicals except arsenic, absorption is assumed to be 100%. For arsenic in soil, the value is 60%.

* Combined Soil Ingestion & Dermal Exposure RBCsnon-cancer= HQ / [(1/RfDadministered x SIF soil ing x ABS)+(1/RfDabsorbed x SIF soil dermal x AF x ABSd)]

APPENDIX F

Chemical Toxicity Profiles

DRAFT FINAL SCREENING LEVEL HHRA
SPOKANE RIVER, WASHINGTON
Coeur d'Alene Basin RI/FS
RAC, EPA Region 10
Work Assignment No. 027-RJ-CO-102Q

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APPENDIX F
Chemical Toxicity Profiles

This appendix contains the chemical toxicity profiles for each of the metals of concern. The profiles summarize the toxic effects of the chemicals of concern along with the toxicity criteria used in the risk assessment for assessing noncancer and cancer effects.

Antimony

Adverse Health Effects of Antimony (Sb; CAS# 7440-36-0)

The comprehensive review of antimony toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1992 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to antimony is based on information provided in the U.S. Environmental Protection Agency [EPA] Integrated Risk Information System [IRIS].

Antimony compounds have been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion, producing effects both in the respiratory and gastrointestinal tracts. Certain antimony compounds may be toxic to the heart. Oral exposure to antimony has been associated with changes in blood chemistry in laboratory animals. There is inconclusive evidence of a relationship between inhalation of antimony trioxide and excess risk of lung cancer (Hathaway et al., 1991).

Elemental antimony is a silvery-white soft metal. It is found at low concentrations in soil, generally 1 part per million (ppm) or lower. The geochemical properties of antimony are similar to those of arsenic (antimony has +3 and +5 valence states). As with arsenic, antimony may be associated with nonferrous ore deposits, and therefore can be a pollutant in industrial environments (Kabata-Pendias and Pendias, 1992). Antimony is a constituent in alloys with nonferrous metals such as tin, lead, and copper. Antimony sulfides are used in the production of rubber and pyrotechnics. Antimony chlorides are used as coloring agents and catalysts. Antimony fluorides are used in organic synthesis and pottery manufacture (Hathaway et. al., 1991).

Pharmacokinetics

Absorption

Antimony is poorly absorbed from the gastrointestinal tract. Gastrointestinal absorption of more soluble forms (antimony tartrate and antimony chloride) in laboratory animals ranges from 2 to 7 percent. For the inhalation route, quantitative information about absorption rate is not available. Although elevated blood and urinary concentrations have been reported in workers exposed to antimony it is uncertain whether absorption was by the inhalation route or by ingestion of inhaled antimony that was cleared from the respiratory tract. Respiratory absorption has, however, been shown to be a function of particle size (ATSDR, 1992). Certain inhaled antimony compounds appear to be retained in the lung for long periods (NLM/HSDB, 2000). No studies were located regarding dermal absorption of antimony in humans, although studies with rabbits suggest that at least some forms of antimony can be absorbed through the skin (ATSDR, 1992).

The issue of bioavailability of antimony is especially important at mining, milling, and smelting sites. This is because the antimony at these sites often exists, at least in part, as a poorly soluble sulfide, and may also occur in particles of inert or insoluble material. These factors all may tend to reduce the bioavailability of antimony.

Distribution and Excretion

The major sites of accumulation for antimony following ingestion are the liver, kidney, bone, skin, and hair. The distribution of antimony may depend upon its valence state in the body. Inhaled trivalent antimony is mainly bound to erythrocytes, while inhaled pentavalent antimony is found in the plasma. Uptake in bone is greater for pentavalent antimony than for trivalent antimony. Absorbed antimony is excreted both through the feces and the urine, however measurements of fecal excretion of absorbed antimony may be complicated by poor gastrointestinal absorption of ingested antimony. Studies in laboratory animals involving the intraperitoneal route of exposure indicate that the valence state of antimony may influence the route of excretion, with pentavalent antimony excreted principally in the urine, and trivalent antimony excreted through the feces. In laboratory animals, excretion of antimony is a two-phase process, consisting of a fast phase where 90 percent of the initial body burden is excreted within 24 hours (the fast phase), and a slow phase with a half-life of 16 days (ATSDR, 1992).

Qualitative Description of Health Effects

Acute Toxicity

Acute ingestion exposure to antimony has produced gastrointestinal effects both in humans and in laboratory animals, with signs and symptoms including pains in the stomach, vomiting and diarrhea. Other than one study noting inflammation in the lungs of rabbits exposed by inhalation to antimony trisulfide, there is no information available regarding toxic effects from acute inhalation exposure to antimony, as a dust or particulate (ATSDR, 1992).

Stibine (antimony hydride) is a colorless gas produced when acid solutions of antimony compounds come into contact with reducing agents. It is a pulmonary irritant and hemolytic agent following short-term exposure in laboratory animals, and it is likely that the same effects would be observed in humans (Hathaway et al., 1991).

Chronic and Subchronic Toxicity

Mild hematological alterations (not specified) and cloudy swelling in the liver were observed in rats administered antimony trioxide orally at 418 mg/kg-day for 24 weeks. Increased serum cholesterol and decreased nonfasting serum glucose levels were observed in rats exposed for a lifetime to 5 ppm potassium antimony tartrate in drinking water. Occupational exposure to high concentrations of antimony trioxide or pentoxide dust (8.87 mg/m³ as antimony) has produced respiratory irritation, including pneumoconiosis, bronchitis, and alteration in pulmonary function (ATSDR, 1992). Symptoms observed in smelter workers exposed to an average concentrations of antimony of 10 mg/m³ (highest exposures were 70 mg/m³) included dermatitis and rhinitis. Less frequent effects included irritation, sore throat, headache, pain or tightness in the chest, metallic taste, nausea, vomiting, diarrhea, and weight loss (Hathaway et al., 1991). Respiratory effects also have been observed in laboratory animals following long-term inhalation of high levels of antimony, including progression from pneumoconiosis to proliferation of alveolar macrophages, interstitial inflammation, and fibrosis (ATSDR, 1992).

Six sudden deaths, two deaths due to chronic heart disease, elevated blood pressure, and abnormal EKG readings were reported in 125 abrasive wheel workers exposed to 2 to

3 mg/m³ antimony in air as antimony trisulfide for up to 2 years (ATSDR, 1992; Hathaway et al., 1991). Inhalation of antimony trisulfide has produced myocardial effects (degenerative changes in the myocardium and altered EKG readings) in some animal studies but not others (ATSDR, 1992).

Reproductive or Developmental Toxicity

An increased incidence of spontaneous abortions and altered menstrual cycles, compared to a control group, has been reported in a group of women working in an antimony metallurgical plant (Belyaeva, 1967, as cited in ATSDR, 1992). However, the levels of antimony exposure and presence of other compounds was not reported. Additionally, information was not presented demonstrating that the control group population was comparable to the study group in terms of factors other than antimony exposure (ATSDR, 1992). There are no studies evaluating developmental or reproductive toxicity of antimony in humans from ingestion exposure (ATSDR, 1992).

Reproductive effects including failure to conceive and decreased number of offspring were reported in rats exposed to 209 mg/m³ antimony trioxide in air prior to conception and throughout gestation (Belyaeva, 1967, as cited in ASTDR, 1992). No gross abnormalities were observed in the offspring of rats exposed to antimony trichloride in drinking water.

Mutagenicity and Genotoxicity

There is limited evidence of genotoxicity of antimony in *in vitro* systems, but none in *in vivo* systems. Types of effects reported include gene mutations, chromosomal aberrations, and chromosomal breakage in mammalian cell systems (ATSDR, 1992).

Carcinogenicity

There is no conclusive evidence regarding carcinogenicity of antimony compounds in humans. Antimony trioxide has been identified by the American Conference of Governmental Industrial Hygienists (ACGIH) as a category A2, suspected human carcinogen, based on limited evidence in human populations and sufficient evidence in laboratory animals (Hathaway et al., 1991; ACGIH, 1999). However, inhalation exposure to 8.87 mg/m³ antimony (as either trioxide or pentoxide dusts) did not affect the incidence of cancer in workers exposed from 9 to 31 years (ATSDR, 1992). The International Agency for Research on Cancer (IARC) has classified antimony trioxide as "possibly carcinogenic" in humans.

The U.S. Environmental Protection Agency (USEPA) has not evaluated the carcinogenicity of antimony (USEPA [IRIS], 2000). There is conflicting evidence regarding the carcinogenicity of antimony in laboratory animals. While increased incidence of lung tumors has been observed in some studies where rats were exposed to antimony trioxide or antimony trisulfide, other studies showed no evidence of excess tumors. The levels of antimony trioxide exposure in which lung tumors were observed in rats were 4 and 36 mg/m³ (Groth et al., 1986; Watt, 1980; Wong et al., 1979, cited in ATSDR, 1992). Antimony trisulfide produced lung tumors in rats when evaluated at a concentration of 17.5 mg/m³ (Groth et al., 1986; Wong et al., 1979, cited in ATSDR, 1992). However, an increased incidence of lung tumors was not observed in rats exposed to 4 mg/m³ antimony trioxide (Biodynamics, 1990, as cited in ATSDR, 1992) or in pigs exposed to 4.2 mg/m³ as antimony trioxide (Watt, 1983, as cited in ATSDR, 1992). It is not known why there are

inconsistencies between these animal studies, though differences in pulmonary retention and clearance related to particle size of the administered antimony compounds may explain the different results. Also, carcinogenicity in the lung may be related to other pulmonary injuries, such as proliferation of alveolar macrophages, inflammation and fibrosis. Differences in the mechanisms of deposition and clearance of particulates between rats and humans may also explain the apparent evidence of carcinogenicity in laboratory animals with the lack of evidence in humans (ATSDR, 1992).

Exposure Route Considerations

Oral

Antimony is poorly absorbed from the gastrointestinal tract. Acute ingestion exposure is irritating to the gastrointestinal tract. Long-term ingestion exposure in laboratory animals may produce abnormal changes in the blood (increased serum cholesterol and decreased nonfasting serum glucose levels).

Inhalation

Inhalation exposure in workers may be associated with effects to the cardiovascular system (elevated blood pressure), and pneumoconiosis and altered pulmonary function. There is no conclusive evidence that antimony is carcinogenic in humans by inhalation, because evidence from studies in human populations is very limited, and carcinogenicity studies in laboratory animals provide conflicting results.

Dermal

No studies were located characterizing toxicity from dermal exposure to antimony compounds.

Sensitive Populations

No studies were located describing particular sensitivities to antimony exposure. Based on the available information, it is possible to infer that individuals with pre-existing pulmonary disease would be more sensitive to inhalation of antimony under workplace conditions.

Toxicity Factors Derived for Risk Assessment

The oral reference dose (RfD) for antimony is based on decreases in nonfasting blood glucose levels, altered cholesterol levels, and decreased longevity in rats administered 5 ppm antimony in drinking water for life. Since there was only one level of antimony administered, a no observed adverse effect level (NOAEL) was not established in the study. The calculated lowest observed adverse effect level (LOAEL) was 0.35 mg/kg-day. An uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 to convert the LOAEL to a NOAEL) was applied to the LOAEL to obtain an oral RfD of 0.0004 mg/kg-day (USEPA [IRIS], 2000). USEPA's confidence in the oral RfD is reported to be low.

An inhalation reference concentration (RfC) has been developed specifically for antimony trioxide. The RfC is based on the occurrence of chronic interstitial inflammation in the lungs and reduced clearance of inhaled particulates, in rats exposed by inhalation for one year. These data were used in a Benchmark Concentration (BMC) analysis (i.e. pulmonary effects

were grouped as quantal responses, and dose-response was modeled using statistical models). The lower 95 percent confidence limit for the 10 percent relative increase in the probability of pulmonary response was determined to be at 0.87 mg/m³, which was transformed to a human equivalent concentration of 0.074 mg/m³. An uncertainty factor of 300 was applied to this NOAEL as follows: an uncertainty factor of 10 is used for the protection of sensitive human subpopulations; an uncertainty factor of 3 is used for interspecies extrapolation; an uncertainty factor of 3 is applied for data base inadequacies (principally, the lack of reproductive and developmental bioassays); and an additional threefold uncertainty factor was applied to account for a less-than-lifetime exposure duration. These factors were rounded to obtain an uncertainty factor of 300. The resulting RfC (0.074 ÷ 300) is 0.0002 mg/m³ (USEPA [IRIS], 2000).

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Arsenic

Adverse Health Effects of Arsenic (As; CAS# 7440-38-2)

The comprehensive review of arsenic toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1998 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to arsenic is based on information provided in the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS]. Additional discussions of the current basis (i.e. skin cancer) for characterizing cancer risks were drawn from the reports prepared by the National Research Council (NRC, 1999) and USEPA Risk Assessment Forum (USEPA, 1988). Reanalysis of epidemiological data on arsenic exposures has indicated an increased incidence of internal cancers (liver, kidney and bladder) in addition to skin cancer. The papers discussing these more recent findings have been incorporated into this profile.

Key issues associated with assessment of risks from exposure to arsenic at Superfund sites have been addressed in this profile. These issues include bioavailability in certain media (i.e., soil), chemical forms in which arsenic occurs in the environment (inorganic versus organic arsenic), toxicity of different valences and forms of arsenic, and the basis for toxicity factors (the cancer slope factor and the reference dose).

Arsenic has been shown to be toxic to human populations in areas of the world where it is present in naturally elevated levels in groundwater, and in certain occupations such as copper smelting and chemical plant workers. There is good evidence that arsenic is carcinogenic in humans by both oral and inhalation routes, while studies have shown that most laboratory animals are substantially less susceptible to arsenic toxicity than humans (ATSDR, 1998). Therefore, this profile focuses on toxicity information obtained from observations of human populations, and considers animal toxicity data to the extent that data in human populations are unavailable.

Elemental arsenic is a silver-gray metal, however it occurs in rock or soil most often as the sulfide in a variety of complex minerals containing copper, lead, iron, nickel, cobalt, and other metals. Arsenic occurs in the environment principally in the +3 oxidation state (arsenite) or the +5 oxidation state (arsenate) (ATSDR, 1998). When ores containing copper or lead are smelted, arsenic can be released into the air as a fine dust. Arsenic trioxide is the most commercially important form of arsenic, and is produced primarily from flue dust that is generated at copper and lead smelters. Arsenic trioxide is no longer produced in the United States. The principal use of arsenic (as arsenic trioxide) is in wood preservatives with a smaller proportion used in the production of agricultural chemicals such as insecticides, herbicides, algicides, and growth stimulants for plants and animals. The agricultural use of many arsenical pesticides has been phased out in the United States due to concerns about human health risks during production or use. Smaller amounts of arsenic are also used in the production of glass and nonferrous alloys, and in the semiconductor industry.

Pharmacokinetics

Absorption

Both arsenate and arsenite are well absorbed by both the oral and inhalation routes. Absorption by the dermal route is generally quite low. Overall absorption by the inhalation route following particle deposition in the respiratory tract is estimated to be 30 to 60 percent of inhaled arsenic. Studies with laboratory animals and human volunteers indicate that oral absorption of arsenite or arsenate is relatively high (50 to 95 percent of ingested arsenic, depending upon chemical form and species). Oral absorption of less soluble arsenic species, such as sulfides or lead arsenate is lower, around 20 to 30 percent of ingested arsenic (ATSDR, 1998). Studies in rabbits suggest that oral absorption of arsenic from ingestion of contaminated soils are reduced compared to arsenic in solution, although the form of arsenic in the soil, as well as the type of soil, can be assumed to influence the degree of absorption (NRC, 1999). Approximately 80 percent of arsenic in soil (primarily as the less soluble sulfide form) was excreted in the feces, compared with 50 percent excreted when administered as a gavage dose of sodium arsenate (Freeman et al., 1993, as cited in ATSDR, 1998). Arsenic in dust or soil was less 3- to 9-fold less bioavailable than arsenic in solution, depending upon whether bioavailability was based on blood arsenic or urinary excretion of arsenic (ATSDR, 1998).

Good correlations between arsenic in soil and urinary arsenic levels in human receptors were reported at a site where site-specific bioavailability adjustment factors (0.18 to 0.25) were used to account for lower bioavailability of arsenic in soil (Walker and Griffin et al., 1998). In the absence of site-specific data, USEPA Region 10 recommends using a default relative bioavailability factor of 0.6 to account for the decreased absorption of ingested arsenic in soil relative to the absorption of soluble arsenic ingested in water (USEPA, 1997; personal communication, Roseanne Lorenzana, 1998, U.S. EPA Region 10).

Percutaneous absorption of arsenic acid mixed with soil was estimated to be 4.5 percent after 24 hours, as measured in rhesus monkeys (Wester et al., 1993, as cited in ATSDR, 1998).

Distribution

Limited information is available on distribution of arsenic in the body. However the available studies indicate that arsenic is distributed to all tissues of the body following absorption, indicating there is little tendency to accumulate preferentially in any of the internal organs (ATSDR, 1998).

Metabolism and Excretion

The metabolism of arsenic involves oxidation/reduction reactions interconverting arsenate and arsenite and methylation which converts arsenite to monomethyl arsonic acid (MMA) and dimethyl arsinic acid (DMA, or cacodylic acid). Methylation followed by urinary excretion represents a detoxification pathway for arsenic. Combined excretion of methylated and inorganic arsenic accounts for 75 percent of the absorbed dose (ATSDR, 1998). There is some evidence of a maximum level of arsenic that can be detoxified through this mechanism (EPA, 1988). The main site of methylation is the liver where the process is mediated by enzymes using S-adenosylmethionine as a methyl donor. Severe dietary restrictions (dietary protein) reportedly can reduce methylating capacity (ATSDR, 1998; EPA, 1988). Very little of absorbed arsenic is excreted in the feces (ATSDR, 1998).

Qualitative Description of Health Effects

Acute Toxicity

Arsenic is a potent toxicant, with the minimum oral lethal dose in humans ranging from 1 to 3 mg/kg. At high levels of exposure, lethality from arsenic ingestion is usually attributed to cardiopulmonary collapse. Lethal doses in animals are higher than in humans, consistent with the trend that animals are less sensitive to arsenic than humans. Nausea, vomiting and diarrhea are common symptoms in humans following acute high-dose ingestion of inorganic arsenic compounds. The effects are likely due to direct irritation of gastric mucosa. Signs of peripheral neuropathy have been experienced in individuals who have ingested inorganic arsenic. The neuropathy is detected as numbness in the hands and feet, progressing to a painful "pins and needles" sensation. Inhalation of dusts containing inorganic arsenic (principally arsenic trioxide dusts) are irritating to the upper respiratory tract (ATSDR, 1998).

Subchronic and Chronic Toxicity

The most distinguishing adverse effects associated with chronic ingestion of arsenic include skin changes and damage to the vascular system. Severe cases of chronic exposure result in a disorder known as "blackfoot disease", which is a progressive loss of circulation in the extremities ultimately leading to gangrene. Blackfoot disease has been reported in one area of Taiwan with elevated levels of arsenic in drinking water supplies (ATSDR, 1998). The "blackfoot disease endemic area" in Taiwan had arsenic concentrations in well water ranging from 0.01 to 1.82 mg/L (Bates et al., 1992). The localized nature of blackfoot disease may be due to the presence of other substances consumed in drinking water (fluorescent substances) that are possible confounders or have caused synergistic effects (ATSDR, 1998; USEPA, 2000). While blackfoot disease has not been reported elsewhere in the world, other less severe signs of vascular injury (such Reynaud's disease) have been reported in other areas). Hyperkeratosis, hyperpigmentation and skin cancer are also distinguishing adverse effects of arsenic exposure, and have been observed in populations in Taiwan, Mexico, India and Chile who consumed drinking water with high levels of arsenic (greater than 0.2 mg/L) (Smith et al., 1992). Hyperkeratosis and hyperpigmentation appear to be the earliest observable signs of chronic exposure. Epidemiological studies identify a lowest observed adverse effects level (LOAEL) of 0.01 to 0.02 mg/kg-day for skin lesions and a no observed adverse effect level (NOAEL) of 0.0004 to 0.0009 mg/kg-day. Inhalation exposure to arsenic concentrations from 0.1 to 1 mg/m³ also reportedly may lead to hyperkeratosis and hyperpigmentation (ATSDR, 1998).

Reproductive or Developmental Toxicity

Evidence of reproductive or developmental toxicity in humans is limited and inconclusive. The available studies in humans do not provide conclusive evidence that ingestion of arsenic, at the level usually encountered in drinking water, causes developmental toxicity. Studies in laboratory animals suggest that arsenic exhibits developmental toxicity (reduced birth weight, fetal malformations, and increased fetal mortality) at high levels of exposure (20 to 70 mg/kg-day, orally). The data suggest that inorganic arsenic does not pose a significant risk of developmental toxicity except at maternally toxic levels (ATSDR, 1998).

Genotoxicity

Inorganic arsenicals appear to be inactive or weak mutagens, but are capable of producing chromosomal effects (chromosomal aberrations and increased sister chromatid exchange frequency) in test systems. Studies with small human populations have detected increased incidence of chromosomal aberrations in peripheral lymphocytes after inhalation and oral exposure. Arsenic and its metabolites do not appear to directly interact with DNA (ATSDR, 1998).

Carcinogenicity

USEPA has given arsenic a carcinogenicity weight-of-evidence classification of A; human carcinogen. This is based on sufficient evidence in humans, including increased lung cancer mortality observed in human populations exposed through inhalation, increased mortality from internal organ cancers (liver, kidney, lung, and bladder), and an increased incidence of skin cancer observed in populations consuming drinking water high in inorganic arsenic (USEPA [IRIS], 2000). The International Agency for Research on Cancer (IARC) has classified arsenic compounds in Group 1, carcinogenic to humans.

There is clear evidence that oral and inhalation exposure to inorganic arsenic may increase the risk of cancer in humans. Studies of smelter workers and pesticide manufacturing workers populations have all found an association between occupational arsenic exposure and lung cancer mortality. One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer. Observations of arsenical pesticide applicators also suggest an association between arsenic exposure and lung cancer (ATSDR, 1998; NRC, 1999; USEPA [IRIS], 2000).

Several epidemiological studies have demonstrated an association between cancer and ingestion of elevated concentrations of arsenic in drinking water. Studies in Taiwan (in the blackfoot disease endemic area) stratified the exposed population into groups based on drinking water exposure to <0.3 mg/L, 0.3 to 0.6 mg/L, and >0.6 mg/L. EPA estimated average drinking water exposure concentrations of 0.17, 0.47, and 0.8 mg/L for purposes of characterizing exposure/incidence relationships. These concentrations corresponded to approximately 0.6 to 2.8 mg/day arsenic intake, using assumptions for a Taiwanese population (3.5-L/day drinking water intake and a 55-kg body weight). Significantly elevated incidences of skin cancer was observed in these exposed populations. Factors influencing the applicability of the Taiwanese studies for assessing arsenic cancer risks in other exposure settings include uncertainties in estimates of exposure to arsenic and presence of specific environmental factors, such as a low protein diet (USEPA, 1988; ATSDR, 1998). In addition to skin cancer, there are several reports indicating that ingestion of arsenic in drinking water increases the risk of cancer in the liver, bladder and kidney in populations from Taiwan, Argentina, and Chile (Bates et al., 1992; ATSDR, 1998, NRC, 1999). Epidemiological studies of drinking water exposure to arsenic in the U.S. have not shown an increased incidence of cancer, with concentrations ranging from 0.05 to 0.2 mg/L. However, the significance of these findings has been considered limited because of the study designs, and small exposed populations that were relatively mobile and that had access to alternate water supplies (USEPA, 1988; Bates et al., 1992).

Exposure Route Considerations

Ingestion

Food and drinking water are the largest sources of arsenic exposure. Total dietary intake of arsenic in the U.S. averages 0.05 to 0.06 mg/day, using residue data combined with food consumption survey data. Average intake of inorganic arsenic in the U.S. was about 0.01 mg/day. A portion of the arsenic ingested in the diet is in the form of low-toxicity organic arsenic. Meat and grains a have relatively lower fraction of organic arsenic, whereas fish, vegetables and fruits have relatively higher fractions of organic arsenic. Organic arsenical compounds are found to accumulate in fish and shellfish. These derivatives (mainly arsenobetaine and arsenocholine, also referred to as "fish arsenic") have been studied by several researchers and have been found to be essentially nontoxic. Estimates of arsenic intake in drinking water in the U.S. are around 0.005 mg/day, but could be greater (0.01 to 0.1 mg/day) in areas with elevated arsenic concentrations in groundwater. Arsenic intake from drinking water is assumed to be entirely inorganic. Naturally occurring arsenic levels in groundwater in the U.S. average around 1 to 2 parts per-billion (ppb), except for some western states with geological features that are naturally elevated in arsenic. Concentrations of naturally occurring arsenic in groundwater in these areas range from 5 to over 100 ppb. In the U.S., over 350,000 people may drink water containing arsenic concentrations higher than the Maximum Contaminant Level (MCL) of 50 ppb (ATSDR, 1998; USEPA, 1988; Smith et al., 1992).

Studies in laboratory animals suggest that low levels of dietary arsenic may be beneficial or essential. Laboratory animals on arsenic-free diets show decreased growth, decreased weight gain and decreased reproductive success (NRC, 1999). However, no specific biochemical mechanism is known through which arsenic could exert a beneficial effect. If arsenic is beneficial to humans, then the daily requirement probably lies between 0.01 and 0.05 mg/day, which is within the level of total arsenic provided by a normal diet (food and water) (ATSDR, 1998; USEPA, 1988).

Arsenic has typically been associated with adverse effects in human populations when exposed to levels in drinking water exceeding 300 ppb over a long period of time (ATSDR, 1998). Skin lesions (hyperkeratosis and hyperpigmentation, skin cancer, cancer of internal organs, and [in Taiwan] blackfoot disease) are characteristics signs of chronic ingestion exposure to elevated levels of arsenic.

Inhalation

Arsenic concentrations in ambient air in remote areas are within <1 to 3 ng/m^3, and 20 to 30 ng/m^3 in urban areas. Large cities have higher concentrations in air due to emissions from coal-fired power plants. Occupational exposure (principally smelter workers) has been associated with an increased incidence of lung cancer. Occupational exposures associated with cancer effects range from 50 to 300 μg/m^3 in air (ATSDR, 1998).

Dermal

Occupational exposure to arsenic dusts, or arsenical pesticide solutions have been reported to produce dermatitis (ATSDR, 1998)

Sensitive Populations and Indicators of Exposure

Genetic factors and age may distinguish human subpopulations that are sensitive to arsenic exposure, especially in their ability for metabolism. Individuals with impaired capacity to methylate and detoxify arsenic may be at greater risk of adverse effects from arsenic exposure. Therefore, individuals with dietary deficiencies or impaired liver function may be more sensitive to adverse effects from arsenic exposure (ATSDR, 1998). One study in Finland suggests that children have lower arsenic-methylating ability than adults (NRC, 1999).

Effective biomarkers of arsenic exposure include levels measured in the urine, hair or fingernails. Arsenic affects the functioning of several enzymes that may prove useful as biomarkers of potential exposure. Characteristic skin changes (hyperkeratosis and hyperpigmentation) observed through dermatological examination might also provide indicators of exposure (albeit not early).

Toxicity Factors Derived for Risk Assessment

The oral reference dose (RfD) is based on the occurrence of hyperpigmentation and hyperkeratosis, and vascular complications observed in the Taiwanese population ingesting elevated levels of arsenic in drinking water. The NOAEL was calculated to be 0.0008 mg/kg-day. An uncertainty factor of 3 is applied to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals. The oral RfD for arsenic is 0.0003 mg/kg-day. According to USEPA, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.0001 to 0.0008 mg/kg/day. An inhalation RfD or reference concentration (RfC) has not been estimated for arsenic (USEPA [IRIS], 2000).

The oral unit risk factor for estimating excess lifetime cancer risks is based on the incidence of skin cancer observed in Taiwanese population ingesting elevated levels of arsenic in drinking water. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption. It was assumed that skin cancer risk in the U.S. population would be similar to the Taiwanese population. The maximum likelihood estimate (MLE) of skin cancer risk for a 70 kg person drinking 2 L of water per day ranged from 1×10^{-3} to 2×10^{-3} for an arsenic intake of 1 μg/kg-day. Expressed as a single value, the cancer unit risk for drinking water is 5×10^{-5} per (μg/L). Details of the assessment are in U.S. EPA (1988) (USEPA [IRIS], 2000). Using the assumptions of 2-L/day drinking water consumption and 70-kg body weight, this unit risk factor converts to an oral slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$. It should be noted that USEPA's assessment is based on prevalence of skin cancer rather than mortality because the types of skin cancer produced by arsenic are not normally fatal.

The inhalation unit risk factor for estimating excess lifetime cancer risks is the based on the incidence of lung cancer observed in two different populations of smelter workers. The resulting unit risk factor is 4.3×10^{-3} per μg/m³ (USEPA, 1998). Using the assumptions of 20 m³/day inhalation rate and 70 kg body weight, this unit risk factor converts to an inhalation slope factor of 15 $(mg/kg\text{-}day)^{-1}$.

USEPA is currently revising the MCL for arsenic. At the request of USEPA, the National Academy of Sciences (NAS) reviewed the current state of science for estimating risks associated with arsenic in drinking water. In its review, completed in 1999, the NAS recommended lowering the MCL from the current interim drinking water standard of 50 µg/L. This recommendation is based on NAS's assessments of the risks of skin, lung, and bladder cancer from drinking water containing inorganic arsenic. The report quantified the risks from bladder cancer and describes potential risks of cardiovascular effects. USEPA plans to propose a revised MCL in 2000 and issue a final arsenic MCL in 2001 (USEPA, 1999).

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Cadmium

Adverse Health Effects of Cadmium (Cd; CAS# 7440-43-9)

The comprehensive review of cadmium toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1999 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to cadmium is based on information provided in the U.S. Environmental Protection Agency [EPA] Integrated Risk Information System [IRIS].

Cadmium has been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion of cadmium-contaminated food. Inhalation of cadmium dust in certain occupational settings may be associated with an increased incidence of lung cancer. Ingestion of elevated levels of cadmium has resulted in toxicity to the kidney and skeletal system, and may be associated with an elevated incidence of hypertension and cardiovascular disease.

Elemental cadmium is a soft, silver-white metal; however, cadmium is not usually found in the environment as a metal. Cadmium is found in the earth's crust at concentrations of about 1 to 2 parts per million (ppm), primarily in association with zinc ores. Cadmium (as cadmium oxide) is obtained mainly as a by-product during the processing of zinc-bearing ores and also from the refining of lead and copper from sulfide ores. Cadmium is used primarily for the production of nickel-cadmium batteries, in metal plating, and for the production of pigments, plastics, synthetics and metallic alloys (ATSDR, 1999).

Pharmacokinetics

Absorption

Cadmium is poorly absorbed from the gastrointestinal tract. Long-term absorption and retention of cadmium is approximately 5 to 6 percent the amount ingested. Absorption of cadmium from food may be lower than absorption from water or solution (i.e., approximately 2.5 percent). The body stores of iron influence cadmium absorption. Individuals with low iron stores exhibit higher absorption of cadmium. Dietary deficiencies in calcium and protein also enhance cadmium absorption (ASTDR, 1999; USEPA [IRIS], 2000, Goyer, 1991). Absorption of inhaled cadmium is approximately 5 to 20 percent. Absorption of cadmium inhaled in cigarette smoke is higher than absorption of cadmium inhaled in aerosols, as measured in laboratory animals. Dermal absorption of cadmium from solution or soil is very limited (ATSDR, 1999).

The issue of bioavailability of cadmium is especially important at mining, milling, and smelting sites. The cadmium at these sites can often exist, at least in part, as a poorly soluble sulfide, and may also occur in particles of inert or insoluble material. These factors can collectively reduce the bioavailability of cadmium.

Distribution, Metabolism and Excretion

Cadmium is widely distributed in the body following either ingestion or inhalation exposure, with much of the body burden found in the liver and kidney. Cadmium ions circulate in plasma bound to sulfhydryl groups in proteins such as albumin and metallothionein. Binding to metallothionein is thought to reduce the toxicity of cadmium. Following ingestion, fecal excretion is high due to poor gastrointestinal absorption. Most cadmium that has been absorbed, however, is excreted very slowly, with fecal and urinary excretion being about equal (ASTDR, 1999).

Qualitative Description of Health Effects

Acute Toxicity

Oral exposure to cadmium in high concentrations causes severe irritation to the gastrointestinal tract. Common symptoms in humans following ingestion of food or beverages containing high concentrations of cadmium include nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea. The emetic dose has been estimated to be approximately 0.07 mg/kg. Acute inhalation exposure to high concentrations of cadmium oxide fume is intensely irritating to the respiratory tract. Signs and symptoms include irritation, coughing dyspnea, tightness in the chest and flu-like symptoms (ASTDR, 1999).

Chronic/Subchronic Toxicity

Longer-term ingestion exposure to cadmium has resulted in adverse effects in the kidney, the skeletal system, cardiovascular system, and the blood. The kidney is considered to be the main target organ of cadmium toxicity following extended oral or inhalation exposure. Elevated incidences of tubular proteinuria have been found in epidemiologic studies of residents of cadmium-polluted areas in Japan and China, and in studies of workers occupationally exposed to cadmium by inhalation. The effects include increased excretion of proteins, amino acids and sugars in the urine (proteinuria, aminoaciduria and glucosuria), and tubular cell degeneration followed by inflammation and fibrosis. Comparison of measured cadmium levels in the kidney of humans (using *in vivo* neutron activation analysis) with incidence of tubular proteinuria has shown that a critical concentration of 200 μg cadmium/g wet weight in the kidney produces tubular dysfunction in 10 percent of the population (ATSDR, 1999; Goyer, 1991).

Cadmium toxicity affects calcium metabolism. Associated skeletal changes possibly related to calcium loss include bone pain, osteomalacia and osteoporosis. This disorder is known as "itai-itai" (or "ouch-ouch") disease, and has been observed in humans chronically exposed to cadmium in food in Japan (ATSDR, 1999; Goyer, 1991).

Studies in human populations provide conflicting evidence of a relationship between cadmium ingestion or inhalation and high blood pressure. Smoking is a confounding factor in studies of cadmium inhalation exposure, because of the known cardiovascular toxicity of cigarette smoke (although cigarette smoke is itself a significant source of cadmium). Studies with laboratory animals involving oral exposure to cadmium have shown some effects on the cardiovascular system (ASTDR, 1999).

Oral cadmium exposure reduces gastrointestinal intake of iron, which may result in anemia if dietary intake of iron is low. Studies in human populations provide conflicting evidence of a relationship between cadmium ingestion and the occurrence of anemia (ATSDR, 1999).

Kidney toxicity is considered to be the most sensitive effect of cadmium exposure. The no observed adverse effect level (NOAEL) for kidney toxicity from oral exposure to cadmium ranges from 0.002 to 0.01 mg/kg-day. An inhalation NOAEL (corresponding to a 4 percent incidence of proteinuria) is estimated to be approximately 0.017 mg/m³ (ATSDR, 1999; USEPA [IRIS], 2000).

Reproductive or Developmental Toxicity

Available studies of human populations have shown no evidence of a relationship between oral or inhalation exposure to cadmium and reproductive or developmental toxicity. Studies with laboratory animals showed no evidence of adverse reproductive effects associated with inhalation exposure. High oral dose exposures to cadmium have shown evidence of reproductive toxicity in male laboratory animals (testicular damage and prostatic lesions). High oral doses in laboratory animals, ranging from 1 to 20 mg/kg, are fetotoxic, resulting in reduced fetal or pup weights and skeletal malformations (ATSDR, 1999).

Mutagenicity and Genotoxicity

There is conflicting evidence as to whether or not cadmium can cause chromosomal aberrations, either in humans or laboratory animals. Cadmium compounds have been shown to be mutagenic in some bacterial or *in vitro* test systems (ATSDR, 1999).

Carcinogenicity

Neither human nor animal studies provide sufficient evidence to determine whether or not cadmium is carcinogenic to humans from ingestion exposure. Ingestion of cadmium did not appear to be carcinogenic in humans in studies of populations in cadmium-impacted areas in England or Belgium. The geographical distribution of elevated prostate cancer rates was shown to parallel distribution of elevated cadmium concentrations in water, soil or crops in Alberta, Canada. Estimates of cadmium exposures were not performed in any of these studies. No evidence of excess cancer mortality was found in populations in Japan consuming cadmium-contaminated rice. ATSDR states that while there is little evidence of an association between ingestion exposure and increased cancer rates, the statistical power of the available studies to detect an effect is not high. Seven studies in rats and mice in which cadmium salts (acetate, sulfate, and chloride) were administered orally have shown no conclusive evidence of a carcinogenic response (USEPA 2000). While one feeding study exhibited increased incidences of tumors of the prostate, testes and hematopoietic system in rats (Waalkes and Rehm, 1992, as cited in ATSDR, 1999), these results are equivocal since the effects were not found to be dose-related, and some of the tumors were benign.

There is conflicting evidence as to whether cadmium is carcinogenic in humans by inhalation exposure. Prolonged inhalation of cadmium by battery and smelter workers, and workers in a cadmium recovery facility may be associated with increased incidences of lung or prostate cancer in some studies. However, in many cases there are confounding factors such as tobacco smoking and exposure to other carcinogenic metals that prevent making definitive conclusions from these epidemiological studies (ATSDR, 1999; Goyer, 1991). The cohort of workers at the cadmium recovery facility (from Colorado) was reevaluated, with

the analysis controlled for ethnicity and smoking history. This study concluded that there was a significant dose-response relationship (both in terms of duration of exposure and concentration in air) between cadmium exposure and lung cancer mortality. Based on this analysis, the excess lifetime cancer risk associated with the previous OSHA standard (100 μg/m³) would be approximately 50 to 111 lung cancer deaths per 1,000 workers. At the current OSHA standard of 5 μg/m³, the lifetime risk was estimated to be 2.6 to 6 lung cancer deaths per 1,000 workers exposed to cadmium for 45 years (Stayner et al., 1992, as cited in ATSDR, 1999). A parallel analysis of this same cohort of workers, which controlled for arsenic exposure concluded there was no association between cadmium exposure and lung cancer, and that arsenic exposure and cigarette smoking were the major determinants of lung cancer risk (Lamm et al., 1992, as cited in ATSDR, 1999). Further review found limitations with both of these studies (Doll, 1992, as cited in ATSDR, 1999), and more detailed assessment of potential exposures to cadmium and arsenic concluded that it was not possible to state whether or not cadmium was a human carcinogen based on this cohort of workers (Sorahan and Lancashire, 1997).

Studies in laboratory animals provide strong evidence of lung cancer resulting from inhaled cadmium (ATSDR, 1999). The U.S. Environmental Protection Agency has categorized cadmium as a probable human carcinogen by inhalation (Group B1) based on limited evidence in humans and sufficient evidence in laboratory animals (ATSDR, 1999; USEPA [IRIS], 2000). Similarly, the U.S. National Toxicology Program (NTP) has classified certain cadmium compounds as substances reasonably anticipated to be human carcinogens. In addition, the International Agency for Research on Cancer (IARC) has classified cadmium as carcinogenic in humans (ATSDR, 1999).

Pending external review, USEPA (1999) has recommended that cadmium be considered a probable human carcinogen by inhalation exposure. The International Agency for Research on Cancer (IARC) has classified cadmium in Group 2A, probably carcinogenic in humans.

Exposure Route Considerations

Ingestion

Cadmium is poorly absorbed from the gastrointestinal tract, however chronic ingestion exposures in humans have produced adverse effects principally in the kidneys and skeletal system. Ingestion of cadmium may interfere with absorption of dietary iron, and may be related to anemia in some cases. Nutritional deficiencies, particularly those associated with iron, calcium, vitamin D and protein may increase susceptibility to cadmium-related adverse effects. Ingestion of cadmium is not considered to be associated with reproductive toxicity or cancer in humans.

Inhalation

Inhalation exposure to cadmium in humans has been associated with adverse effects to the kidney and possibly with an increased incidence of lung cancer. Cadmium produces lung cancer in laboratory animals following inhalation exposure.

Dermal

There is no specific information about dermal toxicity of cadmium. Cadmium in soil or water appears to be poorly absorbed through the skin (ATSDR, 1999).

Sensitive Populations

Populations potentially sensitive to cadmium have not been studied systematically, however it is possible to infer about potential sensitivities based on the available data. Individuals with poor nutritional status, particularly those with deficiencies in iron and calcium, may experience increased absorption of cadmium from the gastrointestinal tract. Individuals with preexisting kidney damage may experience kidney toxicity at cadmium doses lower than for normal individuals (ATSDR, 1999). Smokers are generally more exposed than nonsmokers.

Indicators of Exposure

Blood cadmium levels are relevant indicators of recent exposure. Urinary cadmium levels are not particularly sensitive to recent exposures, but are relevant indicators of total body burden. When the critical concentration in the kidney is reached, urinary cadmium levels increase sharply due to the release of cadmium from metallothionein in the kidney coupled with decreased renal reabsorption of cadmium. Kidney dysfunction, the most sensitive effect, has been measured by increased levels of solutes (proteins and amino acids) in the urine. Increased urinary excretion of creatinine and metallothionein are additional indicators of kidney dysfunction due to cadmium exposure. Urinary excretion of other proteins and enzymes, while not specific indicators of cadmium-related kidney toxicity, also have been proposed as biomarkers of cadmium-related effects (ATSDR, 1999).

Toxicity Factors Derived for Risk Assessment

The USEPA has recently conducted a toxicological review of cadmium and compounds (USEPA, 1999) in support of a proposed revision of the toxicity factors currently listed in IRIS. However the review is currently under external review and the proposed toxicity factors are not finalized. Both the values currently listed in IRIS and the proposed changes are discussed below.

Toxicity Factors Currently Listed in IRIS

The USEPA recommended two oral reference doses (RfDs) for cadmium, one for cadmium exposure from food and one for cadmium exposure from water. Both RfDs recognize that a concentration of 200 μg/g (wet weight) in the human kidney cortex is the highest renal level not associated with significant proteinuria. A toxicokinetic model was used by USEPA to determine the level of chronic human oral exposure (NOAEL) which results in the critical concentration of cadmium in the kidney of 200 μg/g; the model assumes that 0.01 percent day of the cadmium body burden is eliminated per day (USEPA, 1985, as cited in IRIS). Assuming 2.5 percent absorption of cadmium from food or 5 percent from water, the toxicokinetic model predicts that the NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg/kg-day from water and food, respectively (i.e., the doses corresponding to the 200 μg/g critical kidney concentration). An uncertainty factor of 10 to account for intrahuman variability was applied to these NOAELs to obtain an RfD of 0.0005 mg/kg-day (water) and an RfD of 0.001 mg/kg-day (food) (USEPA [IRIS], 2000). No inhalation RfD or reference concentration (RfC) is currently listed for cadmium.

An inhalation unit risk factor of 1.8 x 10^{-3} (μg/m^3)$^{-1}$ has been estimated from lung cancer incidence in the United States cohort of workers (i.e. from the cadmium recovery facility in Colorado). This corresponds to an inhalation cancer slope factor of 6.3 (mg/kg-day)$^{-1}$.

Quantitative estimates of oral carcinogenicity have not been developed, based on inadequate evidence that cadmium is carcinogenic in humans by the oral route of exposure (USEPA [IRIS], 2000).

Proposed Changes for IRIS

The critical toxic effect proposed for both the oral RfD and inhalation RfC is renal dysfunction as indicated by minimal proteinuria/enzymuria. This critical effect is supported from several cross-sectional population studies, especially by the CadmiBel population study of Buchet et al. (1990, as cited in USEPA, 1999). A toxicokinetic model was used with the data in this study to calculate both a daily oral intake and a continuous air concentration of cadmium that would result in a 10 percent occurrence of minimal enzymuria (the critical effect) in the population at the age of 70. A representative level of dietary cadmium intake was integrated into the toxicokinetic model. The net oral intake (model result minus diet) of 0.0007 mg/kg-day was designated the oral RfD. USEPA (1999) has proposed that one RfD be used for oral exposures to all media (i.e., separate RfDs were not proposed for ingestion of cadmium in food or water). The modeled concentration of cadmium inhaled concomitant with this same representative dietary intake was designated as the inhalation RfC of 0.0007 mg/m^3. For both the RfD and the RfC, alternate contributions of intake from background (and therefore different RfDs and RfCs) are described in USEPA's toxicological review (USEPA, 1999).

Considering the results of occupational inhalation exposures, and using a Poisson regression model on the epidemiology data, USEPA is proposing an inhalation unit risk of 4.4×10^{-3} (μg/m^3)$^{-1}$ (USEPA, 1999).

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Iron

Adverse Health Effects of Iron (Fe; CAS# 7439-89-6)

The primary sources for preparation of this profile were information obtained online from the National Library of Medicine [NLM] Hazardous Substances Data Bank [HSDB], as well as information from the U.S. Environmental Protection Agency [USEPA] National Center for Environmental Assessment (USEPA, 1996), and secondary reviews in the literature. A toxicological profile for iron has not been prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], nor has iron been incorporated into the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS]. The focus of this profile is on key issues associated with risk assessment and toxicity for iron at Superfund sites (i.e. the critical effects considered in developing toxicity values, essential nutritional levels versus toxic levels, interactions with other metals).

Iron is one of the major constituents in the lithosphere (i.e. soil and rock), constituting approximately 5 percent in soil. Oxides and hydroxides of iron that are strongly pigmented determine the color of many soils. The concentration and form of iron is one factor that influences the bioavailability of other trace metals in soil (Kabata-Pendias and Pendias, 1992). Iron is an essential element in human nutrition. In general, the principal toxicological consequences for iron are associated with accidental acute ingestion exposures, chronic overload resulting from hemochromatosis, excess dietary iron or frequent blood transfusions (Goyer, 1991).

The primary use of iron is in alloys with carbon, manganese, chromium, nickel and other elements to form steel (NLM/HSDB, 1999).

Pharmacokinetics

Approximately 2 to 15 percent of ingested iron is absorbed from the gastrointestinal tract. During periods of increased iron need (childhood, pregnancy, blood loss) absorption of iron is increased (Goyer, 1991). No information was identified quantifying absorption of iron from inhalation. However, inhalation of high concentrations of iron results in pulmonary deposition. Long-term inhalation exposure to iron has resulted in mottling of lungs observable in chest X-rays, a condition referred to as siderosis (Hathaway et al., 1991).

The overall disposition of iron (i.e. absorption and distribution) is closely regulated to maintain homeostasis. While oral absorption of iron can vary over short periods, excretion of absorbed iron does not typically vary and is usually only about 0.01 percent of body burden. Total iron in the body normally ranges from 3 to 5 g, while total elimination is about 0.5 mg/day. Approximately 70 percent of the iron in the body is bound to hemoglobin or myoglobin in the blood. Excess iron is bound to the proteins ferritin and hemosiderin, which are synthesized in the liver. Along with the liver, the reticuloendothelial system (the spleen) is a storage site for excess iron (Goyer, 1991). Excretion of iron occurs primarily from the gastrointestinal tract (i.e., in the feces), with smaller amounts in urine and sweat. The normal breakdown of red blood cells and hemoglobin leads to the release of

iron and production of bile pigments. Measurements of fecal excretion of iron, however, may also be complicated by poor gastrointestinal absorption of ingested iron.

The issue of bioavailability of iron is especially important when considering soil exposure pathways. This is because the iron in soil can exist, at least in part, as a poorly soluble salt, and may also occur in particles of inert or insoluble material. These factors all may tend to reduce the bioavailability of iron.

Qualitative Description of Health Effects

While iron is an essential element in human nutrition, there is the potential for adverse health effects principally from ingestion exposure.

Acute Toxicity

Acute toxicity has resulted from the accidental ingestion of iron-containing medications, principally by children eating ferrous sulfate tablets with candy-like coatings (although these occurrences were more prevalent before the widespread use of "child-proof" caps on prescription medicine containers). Severe toxicity can occur following ingestion of more than 0.5 g of iron. Predominant signs of overexposure include ulceration of the gastrointestinal tract with vomiting (including blood), black stools, damage to the liver and kidneys, and metabolic acidosis. Death is thought to occur from renal failure and cirrhosis of the liver (Goyer, 1991).

Chronic/Subchronic Toxicity

The normal dietary intake of iron is estimated to range from 11 to 19 mg/day (USEPA, 1996). Chronic iron toxicity or iron overload (from ingestion exposure) can occur from excessive accumulation of iron in the body. There are three ways in which this could occur. The first is due to abnormal iron absorption from the gastrointestinal tract (i.e., idiopathic hemachromotosis). The second is through excessive dietary intake. The third may occur from regular blood transfusions required for some forms of anemia. In all cases, the body burden can increase to 20 to 40 g, and the excess iron accumulates in the liver, spleen, pancreas, endocrine organs and the heart. Adverse effects may include disturbance of liver function, diabetes mellitus, disturbance of endocrine function and cardiovascular effects. On a cellular level, increased lipid peroxidation occurs, resulting in membrane damage to cellular organelles (Goyer, 1991).

Inhalation of iron oxide fumes or dusts by workers in metals industries results in deposition of iron particulate in the lung. Inhalation of iron oxide fume or dust over long periods can cause a benign pneumoconiosis referred to as siderosis. Inhaled iron oxide does not cause fibrotic changes in the lungs of laboratory animals, and it is thought that the same applies to humans. Occupational inhalation exposures of 6 to 10 years can produce changes in the lung detectable by X-rays. The retained particulates produce X-ray shadows that may be indistinguishable from fibrotic pneumoconiosis. However, the observation of siderosis typically has not been associated with reductions in pulmonary function, even with exposures to concentrations higher than 10 mg/m³. Some loss of pulmonary function has been observed in welders exposed to iron oxide fumes. However welders are exposed to a complex mixture of metallic oxide fumes and irritant gases, and the loss of pulmonary function from these causes should not be confused with benign pneumoconiosis caused by iron oxide (Hathaway et al., 1991).

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

A survey in the United Kingdom of iron overdoses by pregnant women using iron supplementation concluded that there was no correlation between serum iron levels, outcome of pregnancy and birth weights, even with serum iron levels sufficient to cause moderate to severe toxicity (McElhatton et al., 1991; 1993, as cited in DART/ETIC). No reports were found of iron toxicity-induced developmental defects in experimental animals.

Mutagenicity and Genotoxicity

Iron is known to catalyze the production of highly reactive oxygen species, which in turn produce DNA damage in cell free systems. Genotoxic effects of iron nitriloacetic acid (iron-NTA) observed in *in vitro* systems include breakage of DNA strands and increased sister chromatid exchange frequency. The implications for DNA *in vivo*, or for human carcinogenicity are not known, though occupational exposure to iron-NTA is suspected of being carcinogenic (Hartwig et al., 1992 as retrieved from EMIC). Hydrogen peroxide-mediated oxidative DNA damage in iron-loaded liver cells may be potentiated by certain iron chelators, while other chelators exert a protective effect (Cragg et al., 1998 as cited in EMIC).

Carcinogenicity

Although a carcinogenic response from chronic ingestion of inorganic iron has not been reported, iron overload may potentiate other carcinogens. In one study, preneoplastic foci were produced in rat liver with diethylnitrosamine as initiator and partial hepatectomy with 2-acetylaminofluorene as promoter. Two weeks after promotion, the rats were fed 1.25 to 2.5 percent (12,500 to 25,000 mg/kg) dietary carbonyl iron for up to 45 weeks. The conclusion from this study was that dietary iron overload resulted in an increased number of preneoplastic foci but did not enhance the progression of these into hepatocellular carcinomas (Stal et al., 1999 as cited in MEDLINE). Iron dextran, which has limited use as a supplement, has been identified as reasonably anticipated to be a human carcinogen by the U.S. National Toxicology Program (NTP, 1998).

An increased incidence of lung cancer has been observed among hematite miners or iron workers exposed to iron oxide. However there may be concomitant factors explaining the observed cancer incidence, including cigarette smoke, silica dust, radon and polycyclic aromatic hydrocarbons (Hathaway et al., 1991, Goyer, 1991). The U.S. Environmental Protection Agency (USEPA) does not provide a weight of evidence classification for iron.

Exposure Route Considerations

Ingestion

The recommended daily allowance (RDA) for iron is 10 mg for children and males, 15 mg for females and 30 mg for pregnant and lactating women (NRC, 1989). Acute oral doses higher than 0.5 g has produced severe gastrointestinal toxicity. Chronic overexposure to iron can result in iron overload, which has produced adverse effects to the liver and other organs. Iron is not considered to be a human carcinogen (except for iron dextran), however iron may potentiate the effects of other carcinogenic substances. Ingestion of iron supplements is not considered to be associated with developmental toxicity.

Inhalation

Inhalation of high concentrations of iron oxide fume produces a benign pneumoconiosis called siderosis. An increased incidence of lung cancer among some populations of workers exposed to iron oxide fume or dust is more likely associated with other carcinogenic agents, including polycyclic aromatic hydrocarbons and radon.

Dermal

There is no information characterizing adverse effects associated with dermal contact with iron compounds.

Sensitive Populations

There are no known sensitive populations for exposure to iron. However, idiopathic hemochromatosis is thought to have a genetic component (Goyer, 1991).

Toxicity Factors Derived for Risk Assessment

USEPA's IRIS database does not currently provide a reference dose, cancer slope factor, or other toxicological information for iron (USEPA 2000). The USEPA Superfund Technical Support Center has developed a provisional oral reference dose (RfD) for iron. USEPA notes that iron is an essential element and that deriving a risk assessment value for it poses special problems in that the dose-response curve is "U-shaped" (i.e. there is a range of doses necessary to maintain health; doses both above and below that range can result in adverse effects). Thus, the provisional RfD must be protective against deficiency as well as toxicity. A NOEL for chronic iron overload has been estimated using the values for dietary intake and iron status indices taken from the second National Health and Nutrition Examination Survey (NHANES II) data base (STSC, 1999; USEPA, 1996). Looker et al. (1988, as cited in USEPA, 1996) made comparisons of dietary iron intake and biochemical indices of iron status using data from NHANES II. The average intakes of iron ranged from 0.15 to 0.27 mg/kg-day. The serum ferritin levels and percent serum transferrin saturation (both indicators of iron overload) were within the normal range. Thus, intake levels of 0.15 to 0.27 mg/kg-day consumed are both considered sufficient to protect against iron deficiency and insufficient to cause the toxic effects of iron overload.

Using the NOAEL of 0.27 (representing the upperbound value in the range of mean dietary iron intakes, dietary plus supplemental, taken from the NHANES II data base) and dividing by an uncertainty factor of 1 yields the provisional chronic oral RfD of 0.3 mg/kg-day. An uncertainty factor of 1 is supported by the fact that iron is an essential element. In addition, the information used to derive the oral RfD for iron was derived from intake data from over 20,000 individuals aged 6 months to 74 years and humans exert an efficient homeostatic control over iron such that body burdens are kept constant with normal variations in diet. This RfD supplies adequate levels of iron to meet the nutritional requirements of adults and adolescents. It does not supply the RDA to members of the population with greater requirements for shorter-than-lifetime durations, including children and pregnant women. Further, this RfD may not be protective of individuals with inherited disorders of iron metabolism, and could be conservative if applied to exposure scenarios involving forms of iron with low bioavailability (USEPA, 1996).

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Lead

Adverse Health Effects of Lead (Pb; CAS# 7439-92-1)

Recent comprehensive reviews of lead toxicity (National Research Council (NRC), 1993; U.S. Environmental Protection Agency [USEPA], 1998a; Agency for Toxic Substances and Disease Registry [ATSDR], 1999; U.S. Department of Health and Human Resources [DHHS], 1997) were the primary sources of information presented in this profile. To a lesser degree, information from USEPA (1998b) on toxicity of lead at low blood lead (PbB) levels was also used. In addition, recent articles (1996-1999) in the peer-reviewed literature on lead toxicity that were not cited in the above documents were also reviewed. In some cases, the primary sources are internally inconsistent. In these cases, this profile relies on the weight of evidence to draw conclusions. In most cases, the weight of evidence is explicitly discussed in the source documents.

Key issues associated with risk assessment and toxicity for lead at Superfund sites (e.g., oral bioavailability, use of pharmacokinetic models, adverse effects from exposure to low levels of lead for subchronic-chronic durations) have received the greatest emphasis in this profile.

Substantial quantities of both human and animal data are available regarding the toxicity of lead. ATSDR (1999) states that human data are preferred to animal data for assessing the potential health effects from lead exposure to persons living or working near hazardous waste sites or to other populations at risk. Therefore, this profile relies primarily on the human data.

Adverse effects of lead in humans are most typically evaluated in terms of PbB level, as an indicator of internal lead dose. External exposure (e.g., mg lead/kg bw-day or mg lead/m³), as is commonly considered for other chemicals, is a far less accurate indicator of exposure to lead than are PbB levels. Therefore, whenever possible, this profile relates adverse effects to PbB levels rather than to external exposure.

Lead is a soft, bluish-gray metal that melts at 327.4°C (ATSDR, 1999). Lead sulfide, phosphate, and oxides are insoluble or practically insoluble in water; lead chloride is slightly soluble; and lead acetate and nitrate are soluble in water (ATSDR, 1999). Some primary uses of lead in the U.S. are in lead-acid storage batteries, ammunition, bearing metals, brass, bronze, cable covering, extruded products, sheet lead, solder, ceramics, type metal, ballast or weights, pigments, glass, radiation shielding, electronics, tubes or containers, oxides, and gasoline additives (ATSDR, 1999). In 1997, 87 percent of lead use in the U.S. was in the production of lead-acid storage batteries; 7.8 percent was used in metal products; and 5.3 percent was used for miscellaneous applications (Smith, 1998)[1].

Pharmacokinetics

Absorption

Lead absorption is influenced by the route of exposure, the exposure medium, speciation and physiochemical characteristics of lead, and the age and physiological states of the exposed individual.

Inhalation Exposure

Approximately 30-50 percent of particulate airborne lead is deposited in the lower respiratory tract of adult humans (ATSDR, 1999). USEPA (1989)[3] estimated that respiratory deposition of lead in children is 25-45 percent. The extent of deposition of inhaled lead can vary depending on such factors as lead speciation, particle size, and characteristics of the respiratory tract (Fleming, 1998; Spear et al., 1998; USEPA, 1986[1]). Almost all lead deposited in the lower respiratory tract is absorbed (USEPA, 1986; Morrow et al., 1980)[1], while lead deposited in the upper respiratory tract is generally transported to the esophagus, then swallowed.

Oral Exposure

Ingested lead is absorbed primarily in the duodenum (Mushak, 1991)[1]. The extent and rate of oral absorption of lead in humans is influenced by physiological states of the exposed individuals (e.g., age, fasting, nutritional status), physiochemical characteristics of the medium and lead ingested (e.g., type of medium, particle size, mineralogy, and lead solubility and species), and dose (ATSDR, 1999).

There is evidence that the percent absorption of lead in humans and animals decreases as intake of lead increases (Diamond et al., 1998; EPA, 1999b; ATSDR, 1999). Saturable mechanisms for lead absorption has been inferred from measurements of net flux kinetics of lead in perfused intestines of animals (Diamond et al., 1998). In addition, numerous observations of non-linear relationships between PbB concentrations and lead intake in humans provide support for the existence of a saturable absorption mechanism or some other capacity limited process in the distribution of lead in humans. (Diamond et al., 1998). However, pharmacokinetic studies on swine suggest that the non-linearity in the lead dose-blood lead relationship could derive from an effect of lead dose on some aspect of biokinetics of lead other than absorption (Diamond et al., 1998).

Nutritional Status

Gastrointestinal absorption of lead may be influenced by nutritional status. Children who are calcium or iron deficient may absorb more lead and have higher PbB levels (Mahaffey et al., 1986; Mahaffey and Annest, 1986; Marcus and Schwartz, 1987; Ziegler et al., 1978)[1]. Calcium in the diet has been shown to reduce absorption of ingested lead in adults (Blake and Mann, 1983; Heard and Chamberlain, 1982)[1].

Age

Gastrointestinal absorption of lead in young children is much higher than in adults. Children 2 weeks to 8 years of age absorb about 40-50 percent of ingested lead (Alexander et al., 1974; Ziegler et al., 1978)[1]. Non-fasted adults may absorb less than 10 percent of water-soluble lead (USEPA, 1996; O'Flaherty, 1998). No experimental data were available regarding absorption of ingested lead in older children. However, one study provides suggestive evidence that children ages 6-11 years absorb similar amounts of lead as do their mothers (Gulson et al., 1997)[1]. Age-dependent differences in absorption of ingested lead

have also been observed in animals (Forbes and Reina, 1972; Kostial et al., 1978; Pounds et al., 1978)[1], and may reflect physiological differences between immature and mature intestines (USEPA, 1986)[1].

USEPA (1998a), USEPA (1996, Adult Lead Exposure Model [ALEM]), and ATSDR (1999) each reported that absorption of ingested lead may increase during pregnancy. However, direct experimental evidence of increased absorption of lead in humans during pregnancy is not available. Instead, higher lead absorption during pregnancy is postulated based on increased calcium absorption and higher maternal PbB levels during pregnancy.

Fasting vs. Non-Fasting

Gastrointestinal tract status (fasting vs. non-fasting) affects lead absorption. The bioavailability of soluble lead in adults may be less than 10 percent when ingested with a meal, but as high as 60-80 percent when ingested after a fast (Blake, 1976; Blake et al., 1983; Blake and Mann, 1983; Graziano et al., 1995; Heard and Chamberlain, 1982; James et al., 1985; Rabinowitz et al., 1976, 1980)[2]. Fasted adults absorbed an average of 26 percent of lead in soil provided from the Bunker Hill Superfund Site compared to only 2.5 percent by non-fasted adults (Maddaloni et al., 1998). A proposed mechanism for these differences is the presence of certain components of ingesta (e.g., fiber, protein, other inorganics) in the small intestine of non-fasted humans that are known to inhibit absorption of inorganics (Ruoff et al., 1994, 1995; Ruoff, 1995).

Because lead is absorbed in the small intestine, the length of fasting required to affect lead absorption could be approximately equal to the amount of time between ingestion and clearing of the contents from the small intestine. In humans, this can take 12-14 hours. Per this definition, persons in the U.S. are not typically in a fasted state. Experimentally derived absorption rates for lead in non-fasting humans may best reflect bioavailability of lead under the typical (non-fasting) human exposure scenarios evaluated in risk assessment. For example, the absolute bioavailability of soluble lead in pregnant women of 20 percent used in USEPA's Adult Lead Exposure Model was calculated based on an estimate of meal-weighted bioavailability, assuming three meals each day and absolute bioavailability of 10 percent for lead ingested just before or soon after a meal (non-fasted state) and 60 percent for lead ingested at other times of the waking day (fasted state) (USEPA, 1996). In calculating bioavailability, it was assumed that adults are in the non-fasted state for 12 of 16 waking hours.

Exposure Medium

Absorption of lead in soil is generally less than that of soluble lead in water or the diet. USEPA pharmacokinetic models for lead assume that the relative bioavailability of lead in soil is only 60 percent of that for soluble lead in water (USEPA, 1994, USEPA, 1996). Absolute bioavailability of lead from soil is assumed to be 30 percent in children (USEPA, 1994) and 12 percent in pregnant women (USEPA, 1996). Maddaloni et al (1998) reported that non-fasted, non-pregnant adults absorbed an average of 2.5 percent of lead ingested in soil.

Speciation and Physiochemical Characteristics

A number of factors may reduce oral bioavailability of lead in soil relative to that for soluble forms of metals used in toxicity studies. These include adsorption of lead to soil, presence of lead in discrete mineral phases in soil, encapsulation of lead inside of insoluble particles in soil, and larger particle sizes of soil (Chaney, 1989). Site-specific bioavailability values lower

or higher than those assumed in USEPA lead models have been reported for lead in mining waste and weathered siliceous industrial slag (ATSDR, 1999; Freeman et al., 1992, 1994[1], 1996[1]; Dieter et al., 1993; Davis et al., 1997; Polak et al., 1996; USEPA, 1999b). Analysis of lead mineralogy at some sites showed that lead was present in relatively insoluble, discrete mineral phases (e.g., lead phosphate) and was encapsulated inside of particles (e.g., in silicates) (Davis et al., 1993; Davis et al., 1997). These properties of lead-bearing mineral phases and particles inhibited the release of soluble lead in the gastrointestinal tract and decreased its absorption.

USEPA has used an immature swine model to assess relative bioavailability of lead in soil at Superfund sites (LaVelle et al., 1991; Casteel et al., 1997). EPA's Technical Review Workgroup (TRW) for lead states that: "Currently, the juvenile swine model design offers the strongest method to measured site-specific bioavailability [of lead]" (USEPA, 1999b). A summary of results from immature swine model studies at sites impacted by mines and smelters is presented in Table 2-5 in ATSDR (1999). Relative bioavailability of lead in soil ingested by immature swine ranged from 50-82 percent of that of a similar dose of highly water-soluble lead acetate (ATSDR, 1999). USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model assumes a relative bioavailability of lead in soil of 60 percent (USEPA, 1994).

Dermal exposure

Absorption of lead in soil from the skin in humans is not well understood. Approximate 30 percent of lead nitrate was absorbed when applied to forearms of adult volunteers (Stauber et al., 1994). However, lead measured in blood and urine increased only negligibly, suggesting that the lead absorbed through the skin did not enter the systemic circulation or was present in the circulation in a form not bound to erythrocytes. Moore et al. (1980) reported that percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate was negligible and that lead by this route was unlikely to pose a threat to human health.

Most pharmacokinetic models for lead do not evaluate the dermal route of exposure (USEPA, 1994, 1996; O'Flaherty, 1998; Leggett, 1993; Bowers et al., 1994). An exception is California's Leadspread model which assumes an increase in PbB level of 0.00011 µg/dL per µg lead/day based on dermal absorption of only 0.06 percent of lead in soil (California Environmental Protection Agency, Department of Toxic Substances Control [DTSC], 1992, 1999).

Distribution

Absorbed lead enters blood; 99 percent of lead in blood is located in red blood cells (DeSilva, 1981; USEPA, 1986; Everson and Patterson, 1980; Hursh and Suomela, 1968)[1]. Blood lead rapidly exchanges with lead in other soft tissues (e.g., kidney, liver, lungs, brain) (ATSDR, 1999). The average half-life for lead in blood in adults ranges from 28-36 days (Rabinowitz et al., 1976; Griffin et al., 1975)[1] and for lead in soft tissues is about 40 days (Rabinowitz et al., 1976)[3].

In adults, approximately 94 percent of total body burden of lead is found in bones (Barry, 1975)[1]. In children, only about 73 percent of total lead body burden is in bone (Barry, 1975)[1]. There are two physiological compartments for lead in bone: an inert compartment with a half-life of approximately 27 years (Rabinowitz et al., 1976)[1] and a labile compartment in rapid equilibrium with lead in soft tissues and blood (Alessio, 1988; Chettle et al., 1991;

Hryhirczuk et al., 1985; Nilsson et al., 1991; Rabinowitz et al., 1976)[1]. Bone lead stores in adults can contribute approximately 40-70 percent of the lead in blood (Smith et al., 1996)[1].

Bone lead may be mobilized into maternal blood during pregnancy and lactation in humans (Gulson et al., 1998, 1999) and animals (Franklin et al., 1997. Lead in maternal blood is efficiently transported to the fetus. ATSDR (1999) reports that the fetal/maternal PbB ratio is about 0.9, based on maternal and umbilical cord PbB levels at time of delivery (Abdulla et al., 1997, Goyer, 1990; Graziano et al., 1990; Schuhmacher et al., 1996)[1]. USEPA (1996) recommends use of a $PbB_{fetal}/PbB_{maternal}$ ratio of 0.9 for the Adult Lead Exposure Model. Breast milk can also be a significant source of lead to nursing infants (Gulson et al., 1998; Mushak, 1998, 1999).

Excretion

Lead in the gastrointestinal tract that is not absorbed is eliminated in the feces. Absorbed lead that is not retained is eliminated in the urine or excreted in the feces following biliary secretion into the gastrointestinal tract (ATSDR, 1999).

Lead Pharmacokinetic Models

A number of lead pharmacokinetics models are available to predict PbB levels based on lead intake in various exposure media. These include models by USEPA (1994), USEPA (1996), DTSC (1992; 1999), O'Flaherty (1998), Leggett (1993), and Bowers et al. (1994). ATSDR (1999) presents a detailed review of the IEUBK, O'Flaherty, and Leggett models (ATSDR, 1999). In addition, numerous recent publications evaluate various aspects of the models used in predicting PbB levels (Bowers and Cohen, 1998; Carroll and Galindo, 1998; Griffin et al., 1999; Lakind, 1998; Oreskes, 1998; Rabinowitz, 1998; Tsuji and Serl, 1996) and the EPA TRW for lead presented a review of lead pharmacokinetic models during the March, 2000 meeting of the Society of Toxicologists. The EPA (1994, 1996) models are typically used at Superfund sites to evaluate risk posed from exposure of adults or children to environmental lead.

The Integrated Exposure Uptake Biokinetic (IEUBK) multicompartmental model (USEPA, 1994) predicts PbB levels in young children, age 0 through 6 years, based on lead intake from air, diet, dust, lead-based paint, soil, and water (available from: http://www.epa.gov/superfund   /programs/lead/prods.htm#software). The IEUBK model has been shown to predict PbB level distributions reasonably well for children exposed primarily in the home (ATSDR, 1999).

The ALEM (USEPA, 1996) predicts PbB levels of pregnant women exposed to lead in soil/dust at work, and their fetuses. The Bowers et al. (1994) model, which served as a basis for the ALEM, also predicts PbB levels in pregnant women and their fetuses exposed to lead in soil/dust at work. However, the default parameter values used in the two models are different. Bowers and Cohen (1998) reported that parameter values recommended in the Bowers et al. (1994) model were better predictors of measured PbB levels of adults at several Superfund sites, than were parameter values recommended in the ALEM.

California's Leadspread model predicts PbB levels in young children (including pica children) and in adult residents and workers, based on lead in air, soil, water, the diet and homegrown produce (DTSC, 1992, 1999).

The O'Flaherty PBPK model (1998) can be used to estimate PbB levels for fetuses, infants, children, adolescents, and adults (including pregnant women and older adults), based on lead intake in air, diet, dust, lead-based paint, soil, and water. The O'Flaherty model has been shown to accurately predict PbB levels in children and adults, except when lead concentrations are very high (O'Flaherty 1993, 1995)[1]. In addition, the O'Flaherty model can predict short-term peaks in PbB levels in children resulting from subchronic exposure to lead (Lakind, 1998).

The Leggett (1993) multicompartmental model can predict PbB levels for children and adults, based on age-specific estimates of average daily inhalation and ingestion of lead (ATSDR, 1999). The Leggett model has been shown to accurately predict PbB levels in adults exposed to low levels of lead (ATSDR, 1999).

ATSDR (1999) has developed guidance for using environmental lead data and media-specific slope factors to estimate PbB levels. Estimated contributions to PbB from all exposure pathways are summed to yield a total predicted PbB level. ATSDR (1999) states: "[Unpublished] preliminary efforts to test [this model's] predictive power have shown promise" (p D-10).

Qualitative Description of Health Effects

The toxic effects of lead are generally the same regardless of the route of entry. Low level exposure to lead primarily affects the central nervous system, growth and development, vitamin D metabolism, and blood; however, most parts of the body can be damaged by high exposure to lead. The most severe neurological effect of lead is encephalopathy, which can lead to permanent neurological effects and death. At lower levels, lead produces more subtle neurological effects that can also be permanent. High levels of lead can produce anemia in adults and children. Lead has been shown to affect some parameters of heme synthesis at low PbB levels with no apparent threshold.

Other targets of lead include the cardiovascular, gastrointestinal, renal, and reproductive systems. There is uncertainty as to whether there is an association between exposure to lead and chromosomal aberrations and increased risk of cancer in workers. Lead is generally considered to be carcinogenic in animals.

Death

Children

Death can result from acute encephalopathy, which occurs in children at PbB levels as low as 80-100 μg/dL (NRC, 1993; ATSDR, 1999).

Adults

Death can result from acute encephalopathy in adults, which occurs at 100-120 μg/dL (NRC, 1993). ATSDR (1999) also reported that severe encephalopathy and death could occur in adults at PbB levels of 100-120 μg/dL.

Increased mortality rates have been reported in workers chronically exposed to lead, from malignant neoplasm, chronic renal disease, cardiovascular disease, cerebrovascular disease, lung cancer, or renal cancer (Cooper, 1988; Cooper et al., 1985; Fanning, 1988; Michaels et al., 1991; Lundstrom et al., 1997; Cocco et al., 1997)[1]. However, others have found no statistically significant increase in mortality rates from occupational exposure to lead (Gerhardsson

et al., 1986; 1995)[1]. With regard to occupational mortality studies, ATSDR (1999) reported that "the results …… are discrepant, and all the studies have design flaws that limit the validity of conclusions that can be drawn from their results" (p. 264).

Neurological Toxicity

Neurological effects occur in developing fetuses and young children at low PbB levels, without an apparent threshold. The brain has little or no capacity to repair injury cause by lead (Landrigan, 1999). Therefore, some adverse neurological effects of lead may be irreversible.

Children

Encephalopathy can occur in children starting with PbB levels of approximately 80-100 µg/dL (Bradley and Baumgartner, 1958[3]; Gant, 1938[3]; Bradley et al., 1956[3]; National Academy of Science [NAS], 1972[3]; NRC, 1993; Rummo et al., 1979[3]; Smith et al., 1983[3]; USEPA, 1986[3]). The early symptoms of encephalopathy can include irritability, poor attention span, headache, muscular tremor, loss of memory, and hallucinations. As encephalopathy progresses, more severe symptoms can appear, including delirium, convulsions, paralysis, coma, and death (Kumar et al., 1987)[3]. Encephalopathy can produce permanent cognitive impairment in survivors, including retardation and severe behavioral disorders (ATSDR, 1999; NRC, 1993).

NRC (1993) identified PbB level LOELs in children of 70 µg/dL for peripheral neuropathy, 30 µg/dL for slower nerve conduction, <25 µg/dL for decreased reaction time, and <10-15 µg/dL for deficits in neurobehavioral development, electrophysiologic changes, and lower IQ. ATSDR (1999) reported that peripheral neuropathy and reduced motor nerve conduction have been observed in children at PbB levels as low as 20-30 µg/dL (Erenberg et al., 1974; Landrigan et al., 1976; Schwartz et al., 1988; Seto and Freeman, 1964)[1].

ATSDR (1999) reported that neuro-developmental deficits are generally better correlated with PbB levels after birth, than with prenatal maternal or neonatal cord PbB levels. Similarly, NRC (1993) reported that "the findings pertaining to the association between indices of prenatal lead exposure and early development are mixed" and that "there is relative little consistency across the set of prospective studies in terms of the association between indices of prenatal lead exposure and later cognitive functions." For example, studies in Boston, Cincinnati, and Cleveland each reported early developmental delays associated with maternal or cord PbB concentrations, whereas studies in Australia did not find associations between prenatal PbB levels and postnatal indices of development (NRC, 1993). In some studies, associations between prenatal exposure to lead and developmental scores attenuated with increased age of the child (NRC, 1993).

It's clear that postnatal lead exposure can lead to persisting deficits in cognitive function in children. NRC (1993) reports that "there are striking consistencies in inverse associations between PbB levels measured in the first few years post-natally and intellectual performance at ages 6-10 years." NRC (1993) reviewed numerous cross-sectional and prospective studies and reported that most studies suggest a 2- to 4-point IQ deficit for each increase of 10-15 µg/dL in blood lead within the range of 5-35 µg/dL. Schwartz (1992) calculated the IQ decline over the blood lead range of 10-20 µg/dL to be 2.32 points for longitudinal studies and 2.69 points for cross-sectional studies[6].

USEPA (1998a) identified IQ deficit as the neuro-toxicological endpoint to be used to estimate the baseline health risks to young children (ages 1-2 years) from exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil in U.S. housing. Based on an evaluation of three meta-analyses of the relationship between PbB levels and IQ deficit decrements (Schwartz, 1993; Pocock et al., 1994; Schwartz, 1994), USEPA (1998a) concluded that a 1 µg/dL increase in PbB level would result on the average in a loss of 0.257 IQ points. Therefore, a doubling of PbB levels from 10-20 µg/dL would result in a loss of approximately 2.57 IQ points (USEPA, 1998a). USEPA (1998a) used this mathematical relationship between PbB level and IQ deficit to evaluate the baseline health risks to young children (ages 1-2 years) in the nation's housing.

Based on evaluation of a number of meta-analyses and cross-sectional and/or prospective studies (Needleman and Gatsonis, 1990; Pocock et al., 1994; Schwartz, 1994; Winneke et al., 1990; International Programme on Chemical Safety [IPCS], 1995), ATSDR (1999) reached similar conclusions to those of USEPA (1998a) and NRC (1993) regarding PbB levels and IQ deficits in children. ATSDR (1999) concluded that "there appears to be a modest association between indices of lead burden, usually PbB, and global indices of development or neuropsychological functioning, usually IQ." (p. 278). ATSDR (1999) also concluded "a doubling of PbB from 10 to 20 µg/dL is associated with an average IQ loss of 1-3 points" (p. 278) A threshold below which lead does not affect IQ in children has not been identified (ATSDR, 1999).

Various behavioral disorders may also occur in children at approximately 10 µg/dL (NRC, 1993). NRC (1993) states that "the most consistent finding across all studies of the CNS effects of lead in children is the association of increasing exposure with increasing reaction time, which apparently indicates an attention deficit." Children with higher lead burdens are more frequently classified as learning-disabled (NRC, 1993). In addition, PbB levels of 10 µg/dL and above have been associated with increased frequency of reading disability, disordered conduct, and possibly increased risk of criminal and delinquent behavior in adolescent and adult life (Landrigan, 1999)

Robinson et al. (1985)[1] reported a lead-related decrease in hearing acuity for young children. Hearing thresholds increased linearly throughout the range of PbB levels of 6-56 µg/dL. USEPA (1998b) reported that altered nerve conduction in auditory pathways and decreased hearing acuity have been observed in children with low PbB levels (Otto et al., 1985; Schwartz and Otto, 1987). The probability of increased hearing thresholds was associated with increased PbB levels from below 4 µg/dL to over 50 µg/dL (Schwartz and Otto, 1987)[4]. Osman et al. (1999) reported that hearing thresholds in children in Poland increased significantly with increasing PbB levels at all investigated sound frequencies, and that the relationship remained significant at PbB levels less than 10 µg/dL.

Alterations in brain electrical activity have also been observed in children at PbB levels of 10-15 µg/dL or lower (Benignus et al., 1981; Otto et al., 1981; Otto et al., 1982; Otto et al., 1985; Robinson et al., 1985; Winneke and Kraemer, 1984; Baumann et al., 1987)[4]. However, it is not known whether the measured alterations in brain activity represent adverse effects (USEPA, 1998b).

Adults

Encephalopathy can occur in adults at PbB levels as low as 100-120 µg/dL (Kehoe, 1961a[1], 1961b[1], 1961c[1], NRC, 1993; Smith et al., 1938[1]). DHHS (1997) indicated PbB levels in adults greater than 80 µg/dL may cause coma, encephalopathy, or death. Encephalopathy in adults can lead to peripheral polyneuritis involving sensory or motor nerves (NRC, 1993).

Overt neurological signs and decreased scores on neuro-behavioral tests have been observed in adults at PbB levels as low as 40-60 µg/dL (Baker et al., 1979, 1983; Campara et al., 1984; Haenninen et al., 1979; Maizlish et al., 1995; Williamson and Teo, 1986; Zimmerman-Tanaelia et al., 1983)[1]. DHHS (1997) reported that workers with PbB levels as low as 40-50 µg/dL may experience fatigue, irritability, insomnia, headaches, and subtle evidence of mental and intellectual decline (Mantere et al., 1984; Hogstedt et al., 1983). However, other studies have reported no effects on neuro-behavioral function in occupationally-exposed adults with PbB levels of 40-60 µg/dL (Milburn et al., 1976; Ryan et al., 1987)[1].

NRC (1993) identified a LOEL for peripheral nerve dysfunction (slower nerve conduction) in adults of 40 µg/dL. DHHS (1997) reported that some subclinical symptoms observed in adult workers exposed to lead, such as peripheral nerve dysfunction, can represent the early stages of permanent neurologic damage to the central and peripheral nervous system.

Two recent studies have reported an association between PbB levels and decreased neuro-behavioral performance in aging subjects with low PbB levels (mean of about 5 µg/dL) (Muldoon et al., 1996; Payton et al., 1998)[1].

Hematological Toxicity

Lead interferes with heme synthesis and erythrocyte function. Reduction of the heme body pool can lead to adverse effects in several physiological systems. For example, decreased heme synthesis can result in decreased hemoglobin levels in blood; decreased levels of 1,25-dihydroxyvitamin D, a hormone that regulates calcium metabolism; and increased blood levels of δ-aminolevulenic acid (ALA), a potential neurotoxicant (USEPA, 1998a).

Anemia can occur at PbB levels of 20-25 µg/dL and higher in children and 50 µg/dL and higher in adults, both from decreased hemoglobin production and increased red blood cell destruction (NRC, 1993). Increases in urinary coproporphyrin (CP-U) and δ-aminolevulenic acid (ALA-U) can occur in children and adults at PbB levels of around 40 µg/dL (NRC, 1993). Other symptoms of decreased heme synthesis and erythrocyte function may be observed at lower PbB levels. These symptoms include increased blood and plasma ALA; increased erythrocyte protoporphyrin (EP), and decreased erythrocyte δ-aminolevulinic acid dehydrase (ALAD) and pyrimidine-5'-nucleotidase activity. Some of these indicators (e.g., decreased erythrocyte ALAD and pyrimidine-5'-nucleotidase activity) may occur at PbB levels around 10 µg/dL or lower with no apparent threshold.

Children

NRC (1993) identified LOELs in children of 70 µg/dL for frank anemia, 40 µg/dL for increasing CP-U and ALA-U, and 20-25 µg/dL for anemia as indicated by reduced hematocrit. Subtle indicators of interference with heme synthesis and erythrocyte function have been observed in children at lower PbB levels. NRC (1993) identified LOELs in children of 15-20 µg/dL for increases in erythrocyte protoporphyrin and for pyrimide-5'-nucloetidase inhibition, and <10-15 µg/dL for ALA-D inhibition. ALAD activity has been

inversely correlated with PbB levels in the general population over the range of PbB levels of 3-34 µg/dL (Hernberg and Nikkanen, 1970)[1]. Erythrocyte pyrimidine-5'-nucleotidase activity has been inversely correlated with PbB levels in children over the ranges of PbB levels of 7-80 µg/dL (Angle and McIntire, 1978)[1] and <10-72 µg/dL (Angle et al., 1982)[1]. ATSDR (1999) reported PbB level thresholds for decreased ALAD or pyrimidine-5'-nucleotidase activity in children has not been identified.

Adults

NRC (1993) identified a LOEL for adults of 80 µg/dL for frank anemia, but further stated that lead workers' hemoglobin concentration is inversely and strongly correlated with PbB concentrations at a threshold of approximately 50 µg/L. NRC (1993) identified LOELs of 40 µg/dL for increasing CP-U and ALA-U, 25-30 µg/dL for erythrocyte protoporphyrin increase in males, 15-20 µg/dL for erythrocyte protoporphyrin increase in females, and <10 µg/dL for ALA-D inhibition. Decreased ALAD activity has been shown to be correlated with PbB levels in the general population over the entire range of PbB levels of 3-34 µg/dL, with no apparent threshold.

Renal Toxicity

Acute nephropathy can occur during the early stages of high exposure to lead, especially in children. Characteristic effects of acute nephropathy are morphological and functional changes in the proximal tubular epithelial cells (Loghman-Adham, 1997). Morphological changes consist of nuclear inclusion bodies, swelling of mitochondria, and cytomegaly of the proximal tubular epithelial cells (Loghman-Adham, 1997). Functional changes consist of aminoaciduria, glucosuria, phosphaturia, and hypophosphatemia; increased sodium and decreased uric acid excretion; and increased excretion of low molecular weight proteins and enzymes (ATSDR, 1999; Loghman-Adham, 1997). In acute nephropathy, glomerular effects are either minimal or absent (ATSDR, 1990). The symptoms of acute nephropathy are generally reversible (ATSDR, 1999).

NRC (1993) identified a LOEL in adults of 100-120 µg/dL for chronic neuropathy in adults. Characteristics of chronic lead nephropathy include progressive interstitial fibrosis, dilation of tubules and atrophy or hyperplasia of the tubular epithelial cells, few or no inclusion bodies, reduction in glomerular filtration rates, and azotemia. Chronic nephropathy can proceed to renal failure, with associated hypertension, hyperuricemia, and gout (Loghman-Adham, 1997). Renal changes produced by chronic nephropathy are generally irreversible (ATSDR, 1999).

ATSDR (1999) reports that efforts to evaluate the effects of lead on renal function have not been consistent when renal biopsies were not performed to prove conclusively the occurrence of nephropathy., and that "this could partially be explained by the choice of the renal function parameter studied" (p. 270).

Children

NRC (1993) identified a LOEL in children of 80-100 µg/dL for renal effects.

In a group of children with PbB levels of 40-120 µg/dL, Pueschel et al. (1972) found aminoaciduria in those with PbB levels of 50 µg/dL or more. Significant increases in urinary N-acetyl-B-D-glucosaminidase (NAG) were reported in children with a mean PbB level of 34.2 µg/dL; NAG activity in the children increased an average of 14 percent for each

10 µg/dL increase in PbB levels (Verberk et al., 1996)[1]. Because NAG might be a sensitive indicator of early subclinical renal disease, ATSDR (1999) reported that some level of tubular damage could occur in children at PbB levels less than 40 µg/dL.

Adults

NRC (1993) identified a LOEL of 100-120 µg/dL for chronic neuropathy in adults. Based on an evaluation of the human database, ATSDR (1999) concluded that chronic nephropathy in occupationally exposed workers is usually associated with PbB levels ranging from 60 to greater than 100 µg/dL. Loghman-Adham (1997) reported that chronic lead-induced nephropathy may develop in adults when PbB levels exceed a threshold of 60 µg/dL.

It is less clear whether adverse renal effects can occur in adults at lower PbB levels. Loghman-Adham (1997) reported that there is a correlation between low PbB levels (e.g., less than 40 µg/dL) and indicators of early renal dysfunction such as serum creatinine and creatinine clearance and urinary excretion of low molecular weight proteins and lysosomal enzymes (e.g., NAG). As discussed above, similar subtle effects on renal function have been observed in children at PbB levels less than 40 µg/dL (Verberk et al., 1996)[1].

Cardiovascular

Acute exposures to high levels of lead can produce cardiac lesions, electrocardiographic abnormalities, and hemolytic anemia in children and adults (ATSDR, 1999).

NRC (1993) identified a LOEL in adults of 10-15 µg/dL for increases in systolic and diastolic blood pressure in adults, including pregnant women. DHHS (1997) stated that studies conducted in the general population suggest that increased PbB levels are associated with small increases in blood pressure, and that the correlation may extend to PbB levels below 10 µg/dL (Pocock et al., 1988; Pirkle et al., 1985; Hertz-Picciotto and Croft, 1993; Schwartz, 1995).

Essentially all studies in humans have reported positive associations between PbB levels and blood pressure, and most of them have reported significant results (NRC, 1993). In 11 epidemiological studies, moderate changes in blood pressure were observed ranging from approximately –0.25 to –3.5 per PbB level change of 10 to 5 g/dL (NRC, 1993). In addition, numerous studies on rats have reported increased blood pressure associated with PbB level. For example, NRC (1993) reported that blood pressure in rats increased from about 114 to 136 as PbB levels increased from about 4 to 17 g/dL (based on Boscolo and Carmingnani, 1988).

Gastrointestinal Toxicity

Colic is an early symptom of lead poisoning in children and adults, characterized by abdominal pain, constipation, cramps, nausea, vomiting, anorexia, and weight loss (ATSDR, 1999). NRC (1993) identified a LOEL of 80-100 µg/dL for colic and other gastrointestinal effects in children. ATSDR (1999) reports that colic has been observed in children at PbB levels of 60-100 µg/dL and higher (USEPA, 1986; NAS, 1972). Symptoms of colic generally occur in adults at PbB levels of 100-200 µg/dL, although they have occurred in some workers at PbB levels as low as 40-60 µg/dL (Awad et al., 1986; Baker et al., 1979; Haenninen et al., 1979; Holness and Nethercott, 1988; Kumar et al., 1987; Marino et al., 1989; Matte et al., 1989; Muijser et al., 1987; Pagliuca et al., 1990; Pollock and Ibels, 1986; Schneitzer, 1990)[1].

Vitamin D Metabolism

Lead can interfere with the conversion of vitamin D to its hormonal form, 1,25-dihydroxyvitamin D (ATSDR, 1999). These effects of lead on vitamin D metabolism may be mediated via lead-induced inhibition of heme synthesis. Altered vitamin D metabolism can adversely affect maintenance of extra- and intra-cellular calcium homeostasis associated with cell maturation and tooth and bone development.

NRC (1993) identified a LOEL in children of 15-20 µg/dL for impaired vitamin D metabolism. USEPA (1998b) reported that reduction in vitamin D hormone synthesis has been observed in children with PbB levels of at least 10-15 µg/dL (based on Rosen, 1995). Large reductions in 1,25-dihydroxyvitamin D have been reported in children with PbB levels of 33-55 µg/dL (Rosen et al., 1980)[1] and 33-120 µg/dL (Rosen and Chesney, 1983; Rosen et al., 1980)[1]. However, Koo et al. (1991)[1] reported that no effects were observed on vitamin D metabolism in children with PbB levels of 4.8-23.6 µg/dL and adequate amounts of calcium, phosphorus, and vitamin D in their diet. IPCS (1995)[1] reviewed the human database with regard to vitamin D metabolism and concluded that PbB levels below 20 µg/dL do not affect circulating concentrations of 1,25-dihydroxyvitamin D in humans with adequate nutritional status. Children with nutritional deficiencies may represent sensitive members of the population with regard to the effects of lead on vitamin D metabolism.

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

Fetuses

There is no question that exposure to prenatal exposure to lead can adversely effect fetuses. OSHA (1998) has stated: "Children born of parents either one of whom were exposed to excess lead levels are more likely to have birth defects, mental retardation, behavioral disorders, or die during the first year of childhood."

NRC (1993) identified a LOEL for reduced gestational age and birthweight of <10-15 µg/dL. NRC (1993) also states that: "some striking inconsistencies, yet to be explained, characterize the data on the relationship between prenatal lead exposure and fetal growth and maturation. For instance, in the large cohort (N=907) of women residing in Kosovo (Factor-Litvak et al., 1991), no associations were seen between midpregnancy PbB concentrations (ranging up to approximately 55 µg/dL) and either infant birthweight or length of gestation."

ATSDR (1999) reported that some studies reported that birth weight may be reduced as maternal or cord PbB levels increase (Bornschein et al., 1989; Dietrich et al., 1986; 1987; Bellinger et al., 1984; McMichael et al., 1986)[1], while other studies did not find an association between maternal or cord PbB levels and birth weight (Ernhart et al., 1985, 1986; Factor-Litvak et al., 1991; Greene and Ernhart, 1991; Moore et al., 1982; Needleman et al., 1984)[1]. Similarly, while some studies reported that gestational age may be reduced at PbB levels as low as 15 µg/dL (Dietrich et al., 1986; 1987; McMichael et al., 1986; Moore et al., 1982)[1], other studies did not find a significant relationship between PbB levels and gestational age (Bellinger et al., 1984; Factor-Litvak et al., 1991; Needleman et al., 1984)[1].

Regarding teratogenic effects of lead in humans, NRC (1993) states that impairments of CNS and other organ developments in fetuses occur at PbB levels of approximately 10 µg/dL. In

a retrospective study of 4,354 infants, Needleman et al. (1984) found a significant increase in the number of minor anomalies observed per child but no malformation was found to be associated with lead.[6]

Adults

High PbB levels can affect reproduction in human males and females (ATSDR, 1999; USEPA, 1998a; DHHS, 1997). Women occupationally exposed to high levels of lead during pregnancy have an increased rate of miscarriages and stillbirths (Nordstrom et al., 1979[3]; McMichael et al., 1986[3]; Baghurst et al., 1987[3]; Rom, 1976[5]). NRC (1993) identified a LOEL of 60 μg/dL for reproductive effects in adult females.

Potential reproductive effects in women from chronic low-level exposure to lead are less understood (NRC, 1993; ATSDR, 1999; USEPA, 1998a). Several large cohort studies with low PbB levels (average level during pregnancy of 5-20 μg/dL) did not report an association between lead and abortions or stillbirths (NRC, 1993). For men, NRC (1993) identified a LOEL of 50 μg/dL for altered testicular function. ATSDR (1999) and DHHS (1997) each reported that some reproductive effects (e.g., decreased sperm count, abnormal sperm morphology, decreased sperm mobility, hormonal changes) can occur among male workers with PbB levels as low as 30-40 μg/dL (Lancranjan et al., 1975[5]; Alexander et al., 1996[1,5],1998; Braunstein et al., 1978[5]; Ng et al., 1991[5]; Gennart et al., 1992[1]; Lerda, 1992[1]; Lin et al., 1996[1]).

The Occupational Safety and Health Administration (OSHA) has stated that its current standard for PbB level of 50 μg/dL in workers may not be protective for adverse effects in fetuses or reproductive effects in men and women (OSHA, 1991) Instead, OSHA recommends limiting PbB levels to less than 30 μg/dL for men or women who "intend to parent in the near future ..... to minimize adverse reproductive health effects to the parents and developing fetus" (OSHA, 1991). The American Council of Governmental Industrial Hygienists (ACGIH) recommends that PbB levels for a woman in the workplace remain below 30 μg/dL, "to protect her ability to have children that can develop normally" (ACGIH, 1994).

Developmental Effects in Children

There is uncertainty regarding the potential effects of prenatal lead on growth in children postnatally. In the Cincinnati prospective lead study, infants born to women with lead concentrations greater than 8 μg/dL during pregnancy grew at a lower rate than expected if increased lead exposure continued during the first 15 months of life (NRC, 1993). If postnatal exposure was low, the infants grew at a higher than expected rate (NRC, 1993). At 33 months, sustained exposure to PbB levels greater than 20 μg/dL were associated with reduced stature; however, prenatal exposure was no longer associated with reduced stature (NRC, 1993)

Postnatal exposure to lead affects growth in children. NRC (1993) reviewed the available data and concluded that postnatal PbB levels of 10-15 μg/dL in children had impacts on growth rates and attained stature. Schwartz et al. (1986)[6] evaluated the large NHANES II data set with respect to height, weight, and chest circumference as a function of PbB concentration. The three growth milestones in children under 7 years old were significantly and inversely associated with PbB concentrations, and the association was present over the PbB concentration range of 5-35 μg/dL. Frisancho and Ryan (1991)[6] found an inverse

association between PbB level and stature in a cohort of 1,454 5- to 12-year old children in the Hispanic HANES data set. Lauwers et al. (1986)[6] in Belgium reported statistically significant and inverse association among growth indexes and PbB concentration in children up to the age of 8 years. Prospective studies have also confirmed an association of postnatal lead exposure with retarded growth in infants and children (NRC, 1993).

Genotoxicity

There is uncertainty regarding the potential effects of lead on human chromosomes.

USEPA (1998a) reported that increased frequencies of chromosomal aberrations have been observed in some studies of occupationally-exposed workers, (Nordenson et al., 1978; Huang et al., 1988), but that most studies report no such increase in workers (Schmid et al., 1972; O'Riordan and Evans, 1974; Bauchinger et al., 1977; Maki-Paakkanen et al., 1981), or in children (Bauchinger et al., 1977). USEPA (1998a) reported that sister chromatid exchanges may (Grandjean et al., 1983; Leal-Garza et al., 1986; Huang et al., 1988), or may not (Maki-Paakkanen et al., 1981; Dalpra et al., 1983) be increased as a result of lead exposure. ATSDR (1999) reported that result of studies with human lymphocyte cultures exposed *in vitro* to lead acetate were nearly equally divided between positive and negative. Evidence in animal systems is also inconclusive (ATSDR, 1999).

Carcinogenicity

Lead is generally considered to be carcinogenic in animals. Evidence regarding carcinogenicity of lead in humans is generally considered to be inadequate.

As reported in USEPA (1998a), increased risks of kidney cancer (Selevan et al., 1985; Steenland et al., 1992; Cocco et al., 1997), lung cancer (Cooper et al., 1985; Gerhardsson et al., 1986; Anttila et al., 1995; Lundstrom et al., 1997), glioma (Anttila et al., 1996), rectal cancer (Fayerweather et al., 1997), and total malignant neoplasms (Cooper and Gaffey, 1975; Cooper, 1976, 1981; Kang et al., 1980; Cooper et al., 1985; Anttila et al., 1995; Gerhardsson et al., 1995; Lundstrom et al., 1997) have been observed in occupationally exposed workers. However, these studies lack necessary details to adequately assess carcinogenicity (USEPA, 1998a). ATSDR (1999) states: "the data currently available do not support an assessment of the potential carcinogenic risk of lead in humans" (p. 289) Similarly, USEPA (IRIS) (1999a) concludes: "the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure."

The carcinogenicity of lead in animals has been conclusively demonstrated (Azar et al., 1973; Koller et al., 1985; Van Esch and Kroes, 1969)[1]. USEPA (1999a) has recommended against using current cancer data in animals to derive a slope factor for use in human risk assessment, stating that "current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk." Based on the animal data, lead has been classified as a probable human carcinogen (B2) by USEPA (1999a), a possible human carcinogen (Group 2B) by IARC, reasonably be anticipated to be a carcinogen by NTP (1998), and an animal carcinogen by ACGIH (1999).

Exposure Route Considerations

The toxic effects of lead are generally considered to be similar regardless of the route of entry (ATSDR, 1999). There is an extensive database relating health effects in humans to

internal dose (e.g., PbB levels), and relatively few data relating human health effects to exposure-route specific external exposure (e.g., mg/kg-day or m³/day). Therefore, the emphasis in this profile has been to correlate health effects in humans with exposure to lead, using PbB levels as an index of exposure. In some cases (e.g., occupational), PbB levels may reflect lead intake via several routes of exposure.

Numerous data are available in animals relating health effects to external dose (mg/kg-day). ATSDR (1999) summarizes many of these studies. However, ATSDR (1999) recommends against using animal data to quantitate human health hazards from exposure to lead, because animal data on lead toxicity are generally considered less suitable for assessing health effects than are human data. Instead, ATSDR (1999) states that human data are the best basis for assessing the potential health effects from lead exposure to persons living or working near hazardous waste sites or to other populations at risk.

Ingestion

Ingestion is the primary route of exposure for children and other non-occupationally exposed receptors. However, dose-response data based on external ingestion dose (mg/kg-day) in children were not located .

ATSDR (1999) reported that ingestion of 0.02-0.03 mg lead acetate/kg-day by adults for 14 days or less resulted in decreased ALAD (Cools et al., 1976; Stuik, 1974). Ingestion of 0.01-0.02 mg lead acetate/kg-day by adults for subchronic durations (3-7 weeks) resulted in decreased ALAD activity, increased red blood cell (RBC) porphyrin, and increased porphyrin IX in RBCs of adults (Cools et al., 1976; Stuik, 1974)[1].

Inhalation

Adults at work may be exposed to lead via inhalation and ingestion of dust. However, very little dose-response data for workers based on external dose (mg/kg-day or mg/m³) in workers. ATSDR (1999) reported that humans inhaling lead at a concentration of 0.011 mg/m³ had a 47 percent decrease in ALAD activity (Griffin et al., 1975). DHHS (1997) reported that severe damage to the peripheral nervous system has occurred historically from chronic, workplace exposures to lead of two or more times higher than the current U.S. Occupation Safety and Health Administration (OSHA) Permissible Exposure Limits [PEL] (Feldman et al., 1977) and that chronic exposure to lead above the OSHA PEL of 0.050 mg/m³ may result in chronic nephropathy and potentially kidney failure.

Under the OSHA general industry lead standard (29 CFR 1910.1025), the PEL for personal exposure to airborne inorganic lead is 50 micrograms per cubic meter (µg/m³) as an 8-hour time-weighted average (TWA) (OSHA, 1978). OSHA states that maintaining the concentration of airborne particles of lead in the work environment below the PEL represents a preventive measure intended to protect workers from excessive exposure, which OSHA defines as a PbB level of 40 µg/dL. ACGIH (1999) has recommended that worker lead exposures be kept below 50 µg/m (as an 8-hour TWA).

Dermal Contact

ATSDR (1999) reported that no studies were located regarding toxicity of lead in humans or animals specifically from dermal exposure. Dermally applied lead nitrate is rapidly absorbed into the skin, but the toxicology significance is not known.

Sensitive Populations

There is evidence that low PbB levels (e.g., 10-15 µg/dL or lower) can adversely affect development in humans exposed prenatally, postnatally, or both.

The embryo/fetus/neonate may be at increased risk because of transfer of maternal lead that may become mobilized from bone during pregnancy and lactation (Gulson et al., 1998, 1999; Mushak, 1998, 1999). Increased rates of miscarriages and stillbirths have been reported in women exposed to high levels of lead during pregnancy (Nordstrom et al., 1979[3]; McMichael et al., 1986[3]; Baghurst et al., 1987[3]; Rom, 1976[5]). In addition, low levels of lead have been associated in some studies with reduced birth weight and gestational age (NRC, 1993). The developing nervous systems of embryo/fetus/neonate may be particularly sensitive to lead toxicity (Rodier, 1995; DHHS, 1997). However, ATSDR (1999) reported that neurodevelopmental deficits in children are generally better correlated with PbB levels after birth, than with prenatal maternal or neonatal cord PbB levels.

Young children are generally at greater risk than adults for experiencing lead-induced health effects due to their physiological, developmental, and behavioral differences (ATSDR, 1999). In comparison with adults, young children absorb more lead from the gastrointestinal tract, retain more absorbed lead, and have a greater prevalence of nutritional deficiency (e.g., calcium, iron, zinc) which can increase both absorption and toxic affects of lead. In addition, the blood-brain barrier is incompletely developed in young children, which may allow greater transfer of lead to the brain, and the developing nervous system of children is more sensitive to the effects of PbB than that of adults. Young children also ingest much more soil/dust and more water and food per kg body weight and inhale more air per kg body weight than adults.

Some women may be at greater risk from exposure to lead because the conditions of pregnancy, lactation, and osteoporosis may intensify lead mobilization from bone demineralization, which can result in higher PbB levels (Bonithon-Kopp et al., 1986c; Markowitz and Weinburger, 1990; Silbergeld, 1991; Silbergeld et al., 1988; Thompson et al., 1985)[1]. Persons with pre-existing neurological dysfunction or kidney disease can be more sensitive to the effects of lead (ATSDR, 1999).

Indicators of Exposure

Several indices in blood and body tissues are available to serve as sensitive biomarkers for lead exposure and toxicity, including lead in blood, bone, and teeth, and physiological changes associated with the effects of lead on heme synthesis (ATSDR, 1999).

Lead in Blood

PbB levels are the easiest and most commonly used index of lead exposure and toxicity (ATSDR, 1999). The average half-life of lead in blood ranges from 28-36 days (Rabinowitz et al., 1976; Griffin et al., 1975)[1]; thus levels in blood reflect to a certain extent recent exposure (ATSDR, 1999). However, lead in blood exchanges with lead in other tissues including bone. Therefore, to a lesser degree, PbB can also reflect body burden which is more related to long-term exposure to lead (ATSDR, 1999). Although measured less often due to methodological problems, lead concentrations in plasma may be a more relevant index of lead distribution to target tissues such as bone marrow, kidney, and the nervous system than PbB levels (Bergdahl et al., 1997, 1999).

Fetuses and Children

A fetal PbB level of 10 μg/dL was recommended by USEPA (1996) for use in the ALEM, based on the assumption that the PbB level of concern for fetuses is the same as that for children. The National Research Council (NRC) (1993) has supported this PbB level of concern for fetuses. For children, 10 μg/dL is generally accepted as a PbB level of concern (USEPA, 1986[4]; 1990[4]; 1996; 1998a; 1998b; CDC, 1991[4]; NRC, 1993). The rational for the selection by several government agencies of 10 μg/dL as the PbB level of concern for children is based on weight-of-evidence which indicates that numerous adverse effects may begin to be seen at PbB levels of around 10 μg/dL (as discussed next).

NRC (1993) reported that the adverse effects that occur at around 10 μg/dL in include (1) impairments in cognitive function and initiation of various behavior disorders in young children, and (2) impairments in calcium function and homeostasis in sensitive populations found in relevant organ systems. NRC (1993) also indicated that some of the neurological effects of lead are likely to be permanent.

USEPA (1998b) reported that USEPA's Air Quality Criteria Document for Lead (USEPA, 1986) concluded that for children: "(1) The collective impact of the effects at blood-lead concentrations above 15 μg/dL represents a clear pattern of adverse effects worthy of avoidance; (2) at levels of 10-15 μg/dL there appears to be a convergence of evidence of lead-induced interference with a diverse set of physiological functions and processes, particularly evident in several independent studies showing impaired neurobehavioral function and development; and (3) the available data do not indicate a clear threshold at 10-15 μg/dL, but rather suggest a continuum of health risks approaching the lowest levels measured" (p. 30316 in USEPA [1998b]).

USEPA (1998b) reported that USEPA's Clean Air Scientific Advisory Committee (CASAC) (USEPA, 1990) concluded that (1) various effects starting at blood-lead concentrations around 10-15 μg/dL or even lower in young children "may be argued as becoming biomedically adverse", (2) blood-lead concentrations at or above 10 μg/dL clearly warrant avoidance, especially for the development of adverse human health effects in sensitive populations and (3) "there is no discernible threshold for several lead effects and that biological changes can occur at lower [PbB] levels [than 10 μg/dL]" (p. 30316 in USEPA [1998b]). The SAB proposed setting 10 μg/dL as the maximal safe PbB level in children.

USEPA (1998b) reported that CDC (1991) stated that ''the scientific evidence showing that some adverse effects occur at blood-lead concentrations at least as low as 10 μg/dL in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure'' (p. 30316 in USEPA, 1998b]).

USEPA (1998b) lists various effects that have been observed at PbB levels of at least 10-15 μg/dL, then states: "While it is possible that some of these effects are reversible (e.g., altered heme synthesis), or have unclear medical or functional implications (e.g., altered brain electrical activity), the Agency believes that the collective impact of these effects on diverse physiological functions and organ systems of young children with blood-lead concentrations as low as 10 μg/dL are clearly adverse" (p. 30316). USEPA (1998b) goes on to state that: "USEPA decided not to establish a level lower than 10 μg/dL because the evidence indicates that health effects at lower levels of exposure are less well substantiated, based on a limited number of studies, a limited number of children, and observation of

subtle molecular changes that are not currently thought to be sufficiently significant to warrant national concern." (p. 30317).

With respect to PbB levels in children higher than 10 µg/dL, CDC (1991)[3] has stated that medical examination and environmental investigation and remediation should be done for all children with PbB levels of 20 to 44 µg/dL and that medical treatment, including chelation therapy, is necessary in children if the lead concentration in blood is 45 µg/dl or higher. PbB levels in a child of 70 µg/dL or higher is a medical emergency (CDC, 1991)[3].

Adults

In adults, there is less agreement regarding a single PbB level of concern. For pregnant women, a maternal PbB level of concern may be approximately 10 µg/dL for protection of the fetus (USEPA, 1996, NRC, 1993). In contrast, ACGIH has recommended that worker PbB levels be kept below 30 µg/dL, "to protect [a women's] ability to have children that can develop normally" (ACGIH, 1994; 1999), OSHA has said that women planning to have children should be advised to limit their PbB levels to less than 30 µg/dL (OSHA, 1991), and the World Health Organization (WHO) recommends that PbB levels in women of reproductive age not exceed 30 µg/dL (WHO, 1980)[5]. NRC (1993) has identified a LOEL of approximately 10 µg/dL for increases in systolic and diastolic blood pressure in adults including pregnant women. DHHS and NIOSH define elevated PbB levels among U.S. adults as those higher than 25 µg/dL (DHHS, 1997). DHHS has established a national goal to eliminate, by the year 2000, all occupational lead exposures that result in PbB levels great than 25 µg/dL (DHHS, 1997). Protective PbB levels for workers in states that require monitoring of PbB levels range from 10-40 µg/dL (DHHS, 1997). OSHA defines excessive exposure to lead as PbB levels greater than 40 µg/dL and requires medical removal of workers with PbB levels greater than 50 µg/dL (OSHA, 1978).

The World Health Organization (WHO) recommends that exposed workers be limited to PbB levels of less than 40 µg/dL (WHO, 1980)[5]. ATSDR (1999) states that a PbB level of 50 µg/dL has been determined to be an approximate threshold for the expression of lead toxicity in exposed workers.

Lead in Other Tissues

Lead accumulates in bone throughout a person's life. Therefore, lead in bone is considered a biomarker of cumulative exposure to lead in children and adults (ATSDR, 1999). *In vivo* tibial X-ray fluorescence (XRF) provides a non-invasive means of estimating cumulative lead absorption (ATSDR, 1999). Recent studies suggest that bone lead levels may be better predictors of some adverse effects than PbB levels (Gonzalez-Cossio et al., 1997[1]; Hu et al., 1994[1]; 1996b[1]; 1998); however, additional research is needed to better understand the relationship between bone lead, exposure, and adverse effects.

Lead in deciduous (i.e., "baby teeth") has been used as a biomarker of lead exposure (Needleman et al., 1993, 1996; ATSDR, 1999). Lead in enamel primarily reflects lead exposure that occurs *in utero* and early infancy, prior to tooth eruption, and lead in dentin is thought to reflect exposure that occurs up to the time the teeth are shed or extracted (Gulson, 1996; Gulson and Wilson, 1994; Rabinowitz, 1995; Rabinowitz et al., 1993).

ATSDR (1999) reports that urinary lead is not a useful biomarker for estimating low-level exposure to lead and that it is difficult to accurately measure endogenous lead in hair due to the potential for external surface contamination.

Physiological Changes

Other sensitive indices of lead exposure and toxicity are related to the effects of lead on heme synthesis. Lead can inhibit the enzyme ALAD, which may lead to decreased ALAD activity in erythrocytes and increased ALA activity in plasma and urine (ATSDR, 1999). Decreased ALAD in blood is a sensitive indicator of recent exposure to lead and may occur at PbB levels in the general population below 10 μg/dL with no apparent threshold (ATSDR, 1999). NRC (1993) has identified LOELs of <10 to 15 μg/dL for children and <10 μg/dL for adults for ALAD inhibition. Urinary ALA, which becomes elevated at PbB levels of around 40 μg/dL in adults and children, is not as sensitive an indicator as ALAD (NRC, 1993).

Lead can inhibit the enzyme pyrimidine-5'-nucleotidase, resulting in an increase in pyrimidine nucleotides in red blood cells. Inhibition of erythrocyte pyrimidine-5'-nucleotidase activity may occur at PbB levels in children below 10 μg/dL with no apparent threshold (ATSDR, 1999).

Lead can inhibit the enzyme ferrochelatase that transfers iron from ferritin to protoporphyrin to form heme. Inhibition of ferrochelatase can result in accumulation of erythrocyte protoporphyrin (EP) [also measured as free erythrocytes protoporphyrin (FEP) and erythrocyte ZPP] in erythrocytes. EP becomes elevated at PbB levels of 15-20 μg/dL in children and 15-30 μg/dL in adults (NRC, 1993) and reflects average lead levels during erythropoiesis over the previous 4 months (ATSDR, 1999).CDC (1991)[1] has defined lead toxicity in children as PbB levels greater than 10 μg/dL and EP levels greater than 35 μg/dL. In medical examinations of lead-exposed workers, OSHA requires measurement of PbB and ZPP levels, hemoglobin and hematocrit determinations, red cell indices, and examination of peripheral blood lead smears to evaluate red blood cell morphology (DHHS, 1997).

Toxicity Factors Derived for Risk Assessment

There is currently no oral reference dose (RfD) for ingestion of lead in IRIS (USEPA, 1999a). USEPA's RfD Workgroup has stated that it would be inappropriate to develop an RfD for lead, because some effects of lead (such as changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development) may occur at blood levels so low as to be essentially without a threshold (USEPA, 1999a).

There is currently no oral slope factor for lead. As stated in USEPA (IRIS) (1999a): "Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used."

No inhalation reference concentration (RfC) or inhalation slope factor is available for lead (USEPA, 1999).

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Manganese

Adverse Health Effects of Manganese (Mn; CAS# 7439-96-5)

The comprehensive review of manganese toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1997 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to manganese is based on information provided in the U.S. Environmental Protection Agency [EPA] Integrated Risk Information System [IRIS].

The focus of this profile is on key issues associated with risk assessment and toxicity for manganese at Superfund site (i.e. the critical effects considered in developing toxicity values, essential nutritional levels versus toxic levels, interactions with other metals). The issue of bioavailability of manganese is especially important when considering soil exposure pathways.

Manganese is one of the more abundant trace elements in soil and rock, with concentrations typically ranging from 200 to 3,500 mg/kg. Manganese occurs most commonly as a divalent cation. It is oxidized under atmospheric conditions and is found in the soil principally as oxides and hydroxides in the form of coatings on soil particles. (Kabatas-Pendias and Pendias, 1992). Manganese and its compounds are used in making steel alloys, dry-cell batteries, ceramics, dyes, welding rods, oxidizing agents, fertilizer and animal food additives (Goyer, 1991).

Pharmacokinetics

Absorption of manganese following ingestion ranges from 3 to 5 percent. Dietary iron deficiency appears to lead to increased manganese absorption. Information is not available regarding absorption following inhalation. Manganese is distributed throughout the body, with the highest levels found in the liver, pancreas and kidney. Studies with laboratory animals indicate that inhaled manganese may be preferentially accumulated in the lung and the brain. Ingested manganese that is absorbed is excreted primarily from the intestines via the bile, in the feces. Smaller amounts of absorbed manganese are excreted in the urine. Approximately 60 percent of inhaled manganese is excreted in the feces, and chronic inhalation exposure results in elevated urinary levels of manganese (ATSDR, 1997).

The issue of bioavailability of manganese is especially important when considering soil exposure pathways. This is because the manganese in soil may exist, at least in part, as poorly soluble salts. These factors all may tend to reduce the bioavailability of manganese.

Qualitative Description of Health Effects

Manganese is an essential element in human nutrition, as a cofactor in several enzymatic reactions. When ingested, manganese is considered to be among the least toxic of the trace elements. The adverse health effects from manganese exposure are principally associated with inhalation exposure in workplace settings. Acute inhalation can produce irritation of

the respiratory tract. Chronic inhalation exposure can produce a central nervous system disorder known as manganism.

Acute Toxicity

Reports of adverse effects in humans from excess acute ingestion exposure to manganese are rare. Manganese reportedly has low oral toxicity in laboratory animals. Inhalation of elevated concentrations of manganese compounds in occupational settings can lead to an inflammatory response in the lungs in humans, producing localized edema. Signs and symptoms of lung irritation may include cough, bronchitis, pneumonitis and small losses in pulmonary function (ATSDR, 1997).

Chronic/Subchronic Toxicity

Manganese reduced survival in chronic feeding studies with rats at doses higher than 200 mg/kg-day, with the cause of death attributed to nephrotoxicity and renal failure. Mice appear to be less sensitive to adverse effects from chronic manganese ingestion (ATSDR. 1997).

Reports of human intoxication following ingestion exposures to manganese are not common. However, information suggests that oral exposure to manganese can produce neurological symptoms in some humans. Individuals in aboriginal islander populations near Australia, who were exposed to elevated concentrations of manganese in drinking water have exhibited symptoms including weakness, ataxia, loss of muscle tone and a fixed emotionless face (Kilburn, 1987, as cited in ATSDR, 1997). Data on concentration-response relationships and lack of a suitable control group limit the conclusions from this study. Other factors besides manganese exposure, including genetic factors, dietary deficiencies and alcohol consumption may have been responsible for the observed symptoms (Cawte et al., 1987; 1989, as cited in ATSDR, 1997). Elevated concentrations of manganese in drinking water (1.8 to 2.3 mg/L) reportedly were associated with increased prevalence of neurological signs in the elderly residents in Greek communities. The occurrence of these effects was compared with a control community with low concentrations of manganese in drinking water (Kondakis et al., 1989, as cited in ATSDR, 1997). While limitations with this study prevent drawing conclusions about the relationship between chronic manganism and manganese in drinking water, the results suggest that chronic oral exposure to manganese can lead to neurological changes in humans.

Numerous studies have concluded that chronic inhalation exposure to high concentrations of manganese compounds can lead to a disabling neurological condition resembling Parkinsonism, which is referred to as manganism. Principal signs include tremors, weakness in the legs, staggering gait, behavioral disorders, slurred speech and a fixed facial expression. Levels of exposure associated with manganism are poorly characterized, but may range from 0.14 to 22 mg/m³. The 0.14 mg/m³ value has been identified by ATSDR as an indicator of subtle neurological effects, and is considered a lowest observed adverse effect level (LOAEL) (ATSDR, 1997).

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

No studies were located regarding developmental effects in humans following oral exposure. Elevated levels of manganese ingestion in rats may lead to a slight delay in maturation of the male reproductive system, without effects to sperm morphology or

reproductive function. Impotence and loss of libido are common symptoms of manganism following high-dose inhalation exposure in human males. Impaired male fertility at levels not producing frank manganism has been reported in one study (ATSDR, 1997).

Mutagenicity and Genotoxicity

Manganese may be clastogenic in mice following oral gavage exposure. Manganese is mutagenic in some bacterial test systems, but is not mutagenic in others. Genotoxicity has been observed in *in vitro* test systems with mammalian cells. These studies suggest that manganese has some genotoxic potential, however the data are not adequate to assess genotoxic risk to humans (ATSDR, 1997).

Carcinogenicity

Inhalation exposure in humans has not been associated with an increased incidence of cancer. Intraperitoneal injection of mice has resulted in lung tumors, in one study. Chronic oral exposures to mice and rats in other studies have indicated small increases in pancreatic tumors (in rats) and pituitary tumors (in mice), though these effects were not dose-related (ATSDR, 1997). A bioassay performed by the U.S. National Toxicology Program (NTP) concluded there was equivocal evidence of carcinogenicity based on a small increased incidence of thyroid gland follicular adenoma and a significantly increased incidence of follicular cell hyperplasia (NTP, 1992, as cited in ATSDR, 1997). The U.S. Environmental Protection Agency (USEPA) has categorized manganese as Group D, not classifiable with regard to human carcinogenicity. The International Agency for Research on Cancer (IARC) has not classified manganese.

Exposure Route Considerations

Oral

Manganese is an essential element in human nutrition. Therefore, any quantitative risk assessment for manganese must take into account aspects of both the essentiality and the toxicity of manganese. Daily intake ranges from 2 to 9 mg/day (Goyer, 1991). The Food and Nutrition Board of the National Research Council (NRC, 1989, as cited in USEPA 2000) determined an "estimated safe and adequate daily dietary intake" (ESADDI) of manganese to be 2 to 5 mg/day for adults. Manganese is poorly absorbed following oral exposure. Reports of human intoxication following ingestion exposures are not common. However, some studies suggest that neurological effects may be associated with consumption of drinking water with elevated levels of manganese. Although ingestion exposure studies suggest that manganese may be weakly carcinogenic in laboratory animals, these data are inadequate to support a classification as carcinogenic by USEPA.

Inhalation

Several studies have shown that inhalation of manganese in occupational settings is associated with a neurological disorder known as manganism. The principal signs of manganism include tremors, weakness in the legs, staggering gait, behavioral disorders, slurred speech, and a fixed facial expression.

Dermal

Other than burns resulting from contact with manganese-containing oxidizing agents, no reports were located describing toxic effects following dermal exposure.

Sensitive Populations

Several researchers have observed considerable variability in neurological effects resulting from exposure to manganese. While the reasons for this are not clear, it is thought that there may be wide variability in manganese absorption and excretion among individuals following either inhalation or ingestion. This variability may be due to differences in transferrin saturation from dietary iron or other metals, calcium or protein intake, or levels of alcohol consumption. The very young have received attention as a potentially sensitive group, based on studies in laboratory animals indicating that neonates absorb and retain higher levels of manganese compared with adults. There are indications but no direct evidence that neonates are more sensitive to manganese-induced neurotoxicity than adult animals. The elderly may also be more sensitive to manganese-induced neurological effects. Individuals with poor iron nutritional status may absorb manganese more readily, and individuals with liver dysfunction may have impaired excretion of manganese, compared with normal individuals (ATSDR, 1997).

Indicators of Exposure

Manganese levels in blood and urine can be indicators of exposure. Blood levels are considered a better indicator of body burden, while urinary levels are a better indicator of recent exposure (ATSDR, 1997).

Toxicity Factors Derived for Risk Assessment

Development of the oral reference dose (RfD) for manganese recognizes that disease states in humans have been associated with both deficiencies and excess intakes of manganese. The oral RfD for manganese is set at 10 mg/day (0.14 mg/kg-day) and is based on the upper end of the normal dietary intake rate. This value is considered a no observed adverse effect level (NOAEL) for dietary intake and is not adjusted with an uncertainty factor. USEPA emphasizes that individual requirements for, as well as adverse reactions to manganese may be highly variable. The reference dose is estimated to be an intake for the general population that is not associated with adverse health effects; this is not meant to imply that intakes above the reference dose are necessarily associated with toxicity (USEPA [IRIS], 2000).

The oral RfD was evaluated further for manganese in other media (drinking water or soil) based on the epidemiologic study of manganese in drinking water, performed by Kondakis et al., 1989, (as cited in USEPA [IRIS], 2000). While the results from this study do not allow a quantitative evaluation of dose-response, they raise concerns about possible adverse neurological effects at doses not far from the range of essentially. For assessing exposure to manganese from drinking water or soil, USEPA (2000) recommends a modifying factor of 3, yielding an oral RfD of 0.047 mg manganese/kg-day (0.14 ÷ 3). They list four reasons for using the modifying factor to adjust the oral RfD for soil and water exposure: (1) in fasted individuals there may be increased uptake of manganese from water; (2) the study by Kondakis et al. (1989) raises some concern for possible adverse health effects associated with a lifetime consumption of drinking water containing about 2 mg/L of manganese;

(3) because infants can be fed formula that typically has a much higher concentration of manganese than does human milk, manganese in the water could represent an additional source of intake for infants; and (4) neonates may absorb more manganese from the gastrointestinal tract, may be less able to excrete absorbed manganese, and absorbed manganese may more easily cross their the blood-brain barrier.

For the CDL human health risk assessment, an oral RfD of 0.14 mg/kg-day was used to evaluate ingestion of manganese in soil, as recommended by USEPA Region 10.

The inhalation reference concentration (RfC) is based a LOAEL for neurological effects of 0.15 mg/m³ based on an 8-hour time-weighted average, for 5 days/week, observed in studies of occupational exposure to manganese dust. This value is converted to a human equivalent concentration (HEC) of 0.05 mg/m³ using USEPA methods for calculating RfCs. An uncertainty factor of 1000 was applied to this value, which reflects a factor of 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental data, providing a RfC of 0.00005 mg/m³ (USEPA [IRIS], 2000). This corresponds to an inhalation RfD of 0.000014 mg/kg-day

The oral RfDs of 0.047 to 0.14 mg/kg-day and inhalation RfD of 0.000014 mg/kg-day for manganese (USEPA 2000) suggest that inhaled manganese may be much more toxic than ingested manganese. Differences in absorption between the two routes cannot alone account for this very large difference. USEPA reports that after absorption via the respiratory tract into blood, manganese is transported through the blood stream directly to the brain, bypassing the liver and first-pass hepatic clearance. They state that this pathway from the respiratory tract to the brain is the primary reason for the differential toxicity between inhaled and ingested manganese. In addition, recent studies in animals have shown that manganese has a unique ability among metals to be taken up in the brain via olfactory pathways (Tjalve and Henriksson 1997). This process involves direct diffusion of manganese from the nasal cavity into the central nervous system without entering blood, therefore bypassing both the first-pass effects of the liver and the blood-brain barrier (Tjalve and Henriksson 1997). This direct pathway to the central nervous system might account in part for the higher toxicity of inhaled manganese.

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Mercury

Adverse Health Effects of Mercury (Hg; CAS# 7487-94-7)

The comprehensive review of mercury toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1999 forms the primary basis for this profile. Specific discussion about toxicity values used to characterize health risks potentially associated with exposure to mercury is based on information provided in the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS].

Key issues associated with assessment of risks associated with mercury at Superfund sites, including bioavailability in certain media (i.e. soil), chemical forms in which mercury occurs in the environment (inorganic versus organic), toxicity of different valences and forms of mercury, and the basis for health guidance values (the reference dose and minimal risk level), have been addressed in this profile.

Mercury has been shown to be toxic to human populations as a result of occupational exposure and accidental ingestion of mercury-contaminated food. The nature of mercury toxicity differs with the chemical form. Elemental mercury vapor and organic mercury vapor have produced toxicity to the central nervous system and kidneys following inhalation exposure in workers. Ingestion of inorganic mercury salts in laboratory animals also has produced toxicity in the kidney. Accidental ingestion exposure to high levels of organic mercury compounds has produced developmental toxicity in humans.

Elemental mercury is a silvery metallic liquid that is volatile at room temperature. Mercury, found in soil and rocks, typically occurs as an ore known as cinnabar, consisting or insoluble mercuric sulfide. Concentrations in soil and rock average 0.5 parts per million (ppm), though actual concentrations vary considerably depending upon location. Mercury is recovered by heating cinnabar and condensing the vapor to form elemental mercury. Much of the mercury produced in the United States comes from secondary sources, such as recycling. The largest use of mercury is for electrolytic production of chlorine and caustic soda. Other uses include electrical devices, switches and batteries, measuring and control instruments, medical and dental applications, and electric lighting.

Pharmacokinetics

Absorption

Absorption following inhalation of elemental mercury vapor is relatively high (74 to 80 percent), however gastrointestinal absorption of elemental mercury is low. Following ingestion, organic mercury compounds are absorbed more readily than inorganic mercury. Animal studies indicate that gastrointestinal absorption of inorganic mercury (as mercuric chloride) ranges from 10 to 30 percent. Absorption of organic mercury compounds following ingestion is very high, with absorption from aqueous solutions being nearly 100 percent. However, bioavailability of methylmercury compounds in some foods (particularly grains) has been shown to be lower compared with aqueous solutions. Although organic mercury compounds (particularly dialkyl mercury) in solution may be readily absorbed

through the skin, elemental and inorganic mercury compounds are not absorbed well dermally (ATSDR, 1999).

The issue of bioavailability of mercury is especially important at mining, milling, and smelting sites. This is because the mercury at these sites often exists, at least in part, as a poorly soluble sulfide, and may also occur in particles of inert or insoluble material. These factors all may tend to reduce the bioavailability of mercury from soil.

Distribution, Metabolism, and Excretion

Once absorbed, both elemental mercury and organic mercury compounds distribute throughout the body. Due to their high lipophilicity, they can readily cross blood-brain and placental barriers. The kidney is a major organ for deposition of both elemental and methyl mercury. Inorganic salts of mercury also distribute throughout the body following ingestion, with highest levels found in the liver and kidney and lowest levels in the brain. Mercuric ion does not readily pass the blood-brain or placental barriers. Elemental mercury is oxidized to the divalent inorganic cation (mercuric ion) principally in the liver, although there is limited evidence that mercuric ion can be reduced to elemental mercury, and excreted by inhalation. Organic mercury is demethylated in the liver to form inorganic mercuric ion. Inorganic mercury is excreted through both the urinary and fecal (biliary) routes, whereas organic mercury compounds are principally excreted through the fecal (biliary) route. Elemental mercury is also excreted by exhalation from the lungs (ATSDR, 1999).

Qualitative Description of Health Effects

Acute Toxicity

Acute inhalation exposure to high concentrations of elemental or organic mercury compounds has occurred under occupational or accidental conditions, producing effects to the respiratory tract (dyspnea, tightness and pains in the chest), cardiovascular system (elevation in heart rate and blood pressure) and gastrointestinal tract (stomatitis, anorexia, bleeding from the gums). Acute oral exposure to inorganic or organic mercury compounds are also associated with cardiovascular and gastrointestinal effects.

Subchronic and Chronic Toxicity

The nervous system is the most sensitive target organ for mercury toxicity following chronic exposures, but kidney toxicity can be manifested following high doses. Effects to the kidney and nervous system can occur from both long-term inhalation and oral exposures. Workers chronically exposed to mercury vapor have shown evidence of kidney toxicity, including proteinuria, albuminuria, and tubular damage, as evidenced from biopsies of kidney tissue. Kidney toxicity has been observed in humans accidentally ingesting inorganic mercury salts. In several studies with laboratory animals, kidney toxicity also has been seen following subchronic and chronic oral exposures to inorganic mercury salts. Evidence of mercury-induced neurological effects comes principally from reported human poisonings from ingestion of methylmercury-contaminated fish in the Minamata area of Japan, and ingestion of seed grain treated with methylmercury fungicides in Iraq. Symptoms reported included ataxia, impaired ability to speak, muscular weakness, abnormal reflexes, mood disorders, distal paresthesias, and impaired hearing and vision (ATSDR, 1999).

Reproductive and Developmental Toxicity

Abortions and decreased litter size are the principal reproductive effects observed in laboratory animals exposed to mercury. Rats administered methylmercuric chloride orally (from 10 to 30 mg/kg) showed increased pre- and postimplantation losses. Maternal body weights were depressed, suggesting that the doses producing reproductive toxicity were maternally toxic. Accidental ingestion of mercury in food has been associated with incidences of developmental toxicity in humans. The large-scale poisonings that occurred in Iraq in 1956, 1960 and 1971-1972, involved ingestion of wheat flour ground from seeds treated with ethylmercury-p-toluene sulfonanilide (a fungicide). Developmental effects included delays in walking and talking, mental retardation and seizures. A dose-response relationship was seen between organic mercury intake and severity of neurological symptoms.

Several ongoing studies of human populations are providing useful information regarding the toxicity of methylmercury. These include studies of populations in the Island of Madeira, Brazil, the Faroe Islands, and the Republic of Seycelles (Risher et al. 1999a). In a cross-section study of the Island of Madeira, 150 first-graders exposed to methylmercury *in utero* and in fish at levels up to 0.8 ppm were evaluated for neurological effects. Mercury levels averaged 14 ppm in the hair of children, and up to 54 ppm in maternal hair (Risher et al. 1999a). Increased latency of the auditory brainstem-evoked potentials in children was found to be related to mercury concentrations in maternal hair. In a cross-sectional study in Brazil, approximately 400 children in 4 villages exposed to mercury *in utero* and in fish were evaluated for neurological effects. Significant correlations were observed between increased mercury concentrations in hair and decreased performance on neurological tests (Risher et al. 1999a). In a study in the Faroe Islands, 917 children, 7 years of age underwent detailed neurobehavioral examination (Grandjean et al. 1997, 1998). Prenatal methylmercury exposure was assessed by measuring maternal hair mercury concentrations. Mild decrements in the domains of motor function, language, and memory were observed in children whose mothers had hair mercury concentrations of 10-20 ppm. The authors concluded that subtle effects on brain function could be detected at "prenatal methyl mercury concentrations currently considered to be safe."(Grandjean et al. 1998). In a prospective longitudinal study in the Republic of Seychelles, children exposed to methylmercury *in utero* and in fish were evaluated. No neurological effects of significance have been detected in this population thus far, in spite of average concentrations of mercury in hair of children of 6.5 ppm (maximum 25.8 ppm) and of mothers of 6.8 ppm (maximum 26.7 ppm) from consumption of an average of 12 fish meals per week (Davidson et al. 1998, as cited in ATSDR, 1999).

Genotoxicity

Mercury may produce chromosomal aberrations in humans and laboratory animals. Studies in human populations consuming methylmercury-contaminated seafood indicates a relationship between exposure and chromosomal breaks in lymphocytes, however the data are limited and considered inconclusive. Studies in rats given high dosages of mercuric chloride by gavage also indicate an dose-related frequency in chromosomal aberrations, including chromatid breaks and unscheduled DNA synthesis (UDS) (ATSDR, 1999).

Carcinogenicity

Results from a 2 year National Toxicology Program bioassay (NTP, 1993, as cited in ATSDR, 1999) indicate that mercuric chloride may cause an increased incidence of thyroid follicular cell tumors and forestomach squamous cell papillomas in rats, and renal carcinomas in mice. Limited animal studies have also shown renal tumors in male rats and male mice following oral exposure to organic mercury. There are no reports describing cancer incidences in human populations exposed to inorganic or organic mercury (dietary exposure or occupational exposure). The U.S. Environmental Protection Agency (USEPA) has classified mercuric chloride and methylmercury into Group C, possible human carcinogens, based on the absence of data in humans and limited evidence of carcinogenicity in animals (USEPA 2000).

Exposure Route Considerations

Ingestion

Adverse effects from ingestion exposure principally have been associated with consumption of grain products or seafood contaminated with organic mercury. The principal adverse effects have been neurological and developmental toxicity. Ingestion of inorganic mercury, the form most likely to be found in soil, has been associated with kidney toxicity in laboratory animals. The adverse effect of concern with soil exposure scenarios therefore is likely to be kidney toxicity. Ingestion studies in laboratory animals exposed to mercury suggest tumor-producing effects.

Inhalation

Adverse effects from inhalation have been associated with occupational exposure to elemental mercury vapor or organic mercury compounds. Accidental poisonings have occurred to children inhaling spilled elemental mercury. The principal adverse effects have been neurological and kidney toxicity. Inhalation toxicity associated with inorganic mercury salts, the form most likely to be found in soil, has not been studied.

Dermal

While organic mercury compounds can be absorbed through the skin, inorganic forms are not expected to be significantly absorbed by this route. Inorganic mercurial compounds used for topical application have produced dermatitis and neurological effects.

Sensitive Populations

Children are considered a sensitive population for exposure to mercury. Differences in sensitivity between children and adults results largely from greater permeability of the underdeveloped blood-brain barrier *in utero* and in infants. Also contributing are differences in routes of exposure and intake rates (for example exposure via ingestion of mothers milk), and importance of developmental milestones during childhood exposure periods (such as language or cognitive development).

In general, young children are exposed to higher doses of methylmercury than are adults (e.g., approximately 1.5- to 2-fold or higher on a body-weight basis). It was recognized that the postnatal nervous system remains vulnerable to methylmercury; however, it is uncertain whether the young child's sensitivity to neurological effects of methylmercury is more like

that of the fetus or that of the adult. Children also appear to have different patterns of tissue distribution of mercury and methylmercury (i.e., biokinetic patterns) than do adults (USEPA, 1999).

Indicators of Exposure

Blood and urinary mercury are typically used as indices of exposure in the workplace. Hair analyses also have been used as indicators of exposure. The most appropriate indicator depends on the form of mercury, the duration of exposure, and time since exposure (ATSDR, 1999).

Toxicity Factors Derived for Risk Assessment

USEPA has published chronic oral reference doses (RfDs) for mercuric chloride and methyl mercury on their Integrated Risk Information System (IRIS) database (USEPA, 2000). The most sensitive adverse effect for mercuric chloride is reported to be formation of mercuric-mercury-induced autoimmune glomerulonephritis. Based on weight of evidence from three subchronic feeding and/or subcutaneous studies in rats, the oral RfD for mercuric chloride is 0.0003 mg/kg-day. All treatment groups exhibited a toxic effect, therefore a no observed adverse effect level (NOAEL) was not reported. An uncertainty factor of 1,000 was applied for extrapolations from LOAEL to NOAEL endpoints, subchronic to chronic exposures, and animal to human populations. USEPA report their confidence in the oral RfD for mercuric chloride is high. A subchronic oral RfD of 0.003 mg/kg-day is provided in the Health Effects Assessment Summary Tables (HEAST) for mercuric chloride, based on autoimmune effects observed in rats from subcutaneous injection (USEPA, 1997).

For methylmercury, the chronic oral RfD in IRIS is 0.0001 mg/kg-day, based on developmental neurologic abnormalities seen in human infants exposed *in utero* due to maternal ingestion of seed grain treated with methylmercury fungicides in Iraq.. Maternal intake levels, estimated based on a concentration of mercury in maternal hair of 11 ppm, were used as the dose surrogate for the observed developmental effects in the infants. An uncertainty factor of 10 was used, and USEPA reported medium confidence in the RfD. A subchronic oral RfD of 0.0001 mg/kg-day is provided in HEAST for methylmercury, based on developmental neurological effects in human infants (USEPA, 1997).

The basis of USEPA's chronic oral RfD of 0.0001 mg/kg-day for methylmercury was described in USEPA's Mercury Study Report to Congress (USEPA 1997). ATSDR (1999) has derived a chronic oral Minimum Risk Level (MRL) of 0.0003 mg/kg-day, based on information from several recent studies of human populations. The MRL was specifically based on the arithmetic mean value of 15.3 ppm mercury in maternal hair during pregnancy for the highest exposed quantile in the 66-month (postnatal) cohort in the Seychelles Child Development Study. Children in this exposure group showed no decrement in performance on neurological tests. An overall uncertainty factor of 4.5 was applied to the NOAEL for mercury exposure to account for potential variability in the U.S. population and possible subtle neurological effects not tested for in the Seychelles Study. Although not identical to the RfD, the ATSDR "safe level" has been reviewed in a number of recent workgroup sessions (Risher et al. 1999a,b), and represents the Department of Health and Human Services official position.

The RfC of 0.0003 mg/m³ is provided in IRIS for elemental mercury vapor, based on neurotoxicity observed in humans and incorporating an uncertainty factor of 30. Occupational studies supporting the RfC reported incidences of hand tremor, increased memory disturbances, and slight autonomic dysfunction. USEPA has reported medium confidence in the RfC for elemental mercury. The RfC is supported by ATSDR's inhalation MRL of 0.002 mg/m³.

No cancer slope factors have been developed for mercury compounds. However, USEPA has classified both mercuric chloride and methylmercury in Group C (possible human carcinogen), based on the absence of data in humans and limited evidence of carcinogenicity in animals whereas elemental mercury is in Group D (not classifiable due to inadequate data) (USEPA 2000).

Recently, USEPA has developed the Mercury Research Strategy to address key scientific questions in order to reduce uncertainties currently limiting the Agency's ability to assess and manage mercury and methylmercury risks (USEPA 1999). This will include evaluations to link toxicity to exposure using a biokinetic model, assessment of sensitive subpopulations, evaluation of recent epidemiological studies, and evaluation of immunological effects.

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Zinc

Adverse Health Effects of Zinc (Zn; CAS# 7440-66-6)

A comprehensive review of zinc toxicity prepared by the Agency for Toxic Substances and Disease Registry [ATSDR], 1994 forms the primary source of information for preparation of this profile. Information regarding the development of toxicity values for zinc has been incorporated from the U.S. Environmental Protection Agency [USEPA] Integrated Risk Information System [IRIS], available online. Additional information regarding uses of zinc and occurrence in the environment has been obtained online from the National Library of Medicine [NLM] Hazardous Substances Data Bank [HSDB].

The focus of this profile is on key issues associated with risk assessment and toxicity for zinc at Superfund sites (i.e. the critical effects considered in developing toxicity values, essential nutritional levels versus toxic levels, interactions with other metals).

Zinc is used primarily in galvanized metals and metal alloys. In addition, various inorganic zinc salts have numerous commercial uses. Zinc oxide is used in the rubber industry as a vulcanization activator and accelerator and to slow down oxidation, and also as a reinforcing agent, heat conductor, pigment, UV stabilizer, supplement in animal feeds and fertilizers, catalyst, chemical intermediate, and mildew inhibitor. Zinc sulfate is used in rayon manufacture, agriculture, zinc plating, and as a chemical intermediate and mordant. Zinc chloride is used in smoke bombs, in cements for metals, in wood preservatives, in flux for soldering; in the manufacture of parchment paper, artificial silk, and glues; as a mordant in printing and dye textiles, and as a deodorant, antiseptic, and astringent. Zinc chromate is used as a pigment in paints, varnishes, and oil colors. Zinc compounds are also used as ingredients in products, such as sun blocks, diaper rash ointments, deodorants, athlete's foot preparations, acne and poison ivy preparations, and antidandruff shampoos (ATSDR, 1994).

Pharmacokinetics

The body's natural homeostatic mechanisms control zinc absorption from the gastrointestinal tract. Persons with adequate nutritional levels of zinc absorb approximately 20 to 30 percent of all ingested zinc. However, zinc-deficient individuals absorb greater proportions of administered zinc. Other differences in zinc absorption are probably due to the type of diet (amount of zinc ingested, amount and kind of food eaten). For example, dietary protein facilitates zinc absorption. High phosphorus intakes in animals decrease zinc absorption, and dairy products that contain both calcium and phosphorus reportedly decrease zinc absorption in humans. Complexing of zinc with amino acids generally enhances zinc absorption (ATSDR, 1994).

Absorption of zinc in the lungs has not been quantitatively studied. Zinc absorption in the lungs is dependent on the compound, particle size, solubility, and the condition of the lungs. Inhaled zinc is also subject to gastrointestinal absorption due to ciliary clearance and

swallowing. Elevated levels of zinc have been found in the blood and urine of workers exposed to zinc oxide fumes (ASTDR, 1994).

Zinc is one of the most abundant trace metals in humans. It is found normally in all tissues and tissue fluids and is a cofactor in over 200 enzyme systems. Together, muscle and bone contain approximately 90 percent of the total amount of zinc in the body. Zinc is present in blood plasma, erythrocytes, leukocytes, and platelets, but is chiefly localized within erythrocytes. Zinc deficiency has been demonstrated to decrease the ability of erythrocytes to resist hemolysis *in vitro*, suggesting that zinc stabilizes erythrocyte membranes. Much of the zinc in plasma is bound to albumin. The limited number of binding sites for zinc in plasma albumin may regulate the amount of zinc retained by the body; albumin-bound zinc has been correlated with plasma zinc levels (ATSDR, 1994).

Zinc is found in blood serum at a concentration of approximately 1 mg/L in both men and women. Several studies have reported increased levels of zinc in the serum and urine of humans and animals after inhalation, oral, or dermal exposure to zinc. However, relationships between serum and/or urine levels and zinc exposure levels have not been established. Excretion of zinc from the body occurs mostly from the intestine, in the feces, although some zinc is also excreted through the kidneys, in the urine. Fecal and urinary excretion of zinc increases as intake increases. Following ingestion, fecal excretion is high due to both poor gastrointestinal absorption and biliary secretion of zinc. Studies with rats confirm that zinc is excreted in the bile (ATSDR, 1994).

Qualitative Description of Health Effects

Zinc is an essential element in human nutrition, required for the proper functioning of numerous metalloenzymes and proper cell growth and division. Zinc deficiency has been associated with dermatitis, anorexia, growth retardation, poor wound healing, impaired reproductive capacity, impaired immune function, and depressed mental function; an increased incidence of congenital malformations in infants has also been associated with zinc deficiency in the mothers (ATSDR, 1994). The recommended daily allowance (RDA) is 15 mg for adult males, 12 mg for adult females, 15 mg for pregnant women, 19 mg for nursing mothers during the first 6 months and 16 mg during the second six months, 10 mg for children older than 1 year, and 5 mg for infants 0 to 12 months old (NRC, 1989, as cited in ASTDR, 1994). Excessive exposure to zinc is reported to be relatively uncommon, and requires high levels of exposure. Zinc does not accumulate in the body with continued exposure, and levels in the body are modulated by homeostatic mechanisms (Goyer, 1991).

Acute Toxicity
Gastrointestinal distress is a common symptom following acute oral exposure to zinc compounds. Accidental poisonings have occurred as a result of the use of zinc supplements and from food contamination caused by the use of zinc galvanized containers. Symptoms develop within 24 hours and include nausea, vomiting, diarrhea, and abdominal cramps (Goyer, 1991) A single dose estimated to be 6.7 mg/kg ingested in water (limeade prepared in a galvanized container) produced gastrointestinal distress and diarrhea. Vomiting, abdominal cramps, and diarrhea with blood was observed in one individual after ingestion of 440 mg zinc sulfate/day (2.6 mg zinc/kg-day) in capsules as a medically prescribed treatment. Gastrointestinal upset (abdominal cramps, vomiting, nausea) occurred in 26 of 47 healthy volunteers following ingestion of zinc sulfate tablets (150 mg as zinc ion in three divided doses per day, 2 mg zinc/kg-day) for 6 weeks. Gastrointestinal effects have also

been observed in laboratory animals, including reduced food consumption and ulceration of the stomach lining.

Acute oral exposure to 2.6 mg zinc/kg-day as zinc sulfate for 1 week resulted in anemia in one person, however it was noted that the anemia may have been secondary to the gastrointestinal hemorrhages. Treatment-related changes in hematological parameters have been observed in humans and animals after intermediate or chronic exposure to zinc or zinc-containing compounds.

Inhalation exposure to high concentrations of some zinc compounds (zinc oxide fume) has been associated with "metal fume fever". Attacks of metal fume fever are characterized by chills and fever, weakness, and sweating. Recovery usually occurs within 24 to 48 hours. Exposure of guinea pigs to zinc oxide fumes at a concentration of 5 mg/m^3 (the Threshold Limit Value) 3 hours/day for 6 days produced temporary decrements in lung volume and carbon monoxide diffusing capacity. These functional changes were correlated with increased lung weight, inflammation involving the proximal portion of alveolar ducts and adjacent alveoli, interstitial thickening, and increased pulmonary macrophages and neutrophils in adjacent air spaces (Goyer, 1991; ATSDR, 1994). Zinc chloride, a corrosive inorganic salt, is more damaging than zinc oxide to the mucous membranes of the nasopharynx and respiratory tract upon contact. Zinc chloride is a primary ingredient in smoke bombs used by the military for screening purposes, crowd dispersal, and fire-fighting exercises. Serious respiratory injury has resulted from accidental inhalation of smoke from these bombs (ATSDR, 1994).

Chronic/Subchronic Toxicity

Longer-term administration (1 to 8 years) of zinc supplements (in one case, 2 mg/kg-day as zinc sulfate) has caused anemia in humans. Oral administration of zinc compounds produced decreased hemoglobin, hematocrit, erythrocyte, and/or leukocyte levels in rats. A lowest observed adverse effects level (LOAEL) with subchronic exposure in rats (1 month) was 12 mg/kg-day as zinc chloride. However, a no observed adverse effect level (NOAEL) for hematological effects of 191 mg/kg-day as zinc acetate was reported, following 3 months exposure in rats. The considerable range in effects levels is not clear, but may be due to different zinc compounds or differences in strains or ages of the test animals. Mice appear to be less sensitive to hematological effects from zinc exposure compared to rats.

Exposure to 191 mg/kg-day of zinc acetate administered orally in rats over 3 months produced no liver toxicity, but produced kidney toxicity, with epithelial cell damage in the glomerulus and proximal convoluted tubules and increased plasma creatinine and urea levels. Again, mice appeared to be relatively less sensitive to renal effects from zinc exposure compared to rats (ATSDR, 1994).

Teratogenicity, Reproductive Toxicity, and Fetotoxicity

Little information is available on the developmental and reproductive toxicity of inorganic zinc to humans or animals. Reproductive toxicity observed in laboratory animals (principally rats) includes fetal resorption, increased stillbirths, preimplantation losses and reproductive failure. The lowest observed adverse effects levels for these effects range from 200 to 250 mg/kg-day (ATSDR, 1994). Only one report in the literature suggested adverse developmental effects in humans due to exposure to zinc. Four women were given zinc supplements of 0.6 mg zinc/kg-day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant (Kumar

1976, as cited in ATSDR, 1994). However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming up to 0.3 mg zinc/kg-day (as zinc sulfate) during the last two trimesters of pregnancy (ATSDR, 1994).

Information on the developmental and reproductive toxicity of inorganic zinc compounds to humans by other routes of exposure was not available.

Mutagenicity

Genotoxicity studies have provided very limited evidence of mutagenicity and of weak clastogenic effects. Zinc chloride is reported to be positive in the Salmonella assay, negative in the mouse lymphoma assay, and a weak clastogen in cultured human lymphocytes. Zinc sulfate is reported to be not mutagenic in the Salmonella assay, and zinc acetate is reported to not induce chromosomal aberrations in cultured human lymphocytes. Zinc oxide was not mutagenic in Salmonella (USEPA, 2000). Chromosomal aberrations have been observed in bone marrow cells in rats following exposure to 14.8 mg zinc/kg-day as zinc chlorate in drinking water. An increased incidence of sister chromatid exchange was observed in bone marrow cells with a drinking water dose of 17.5 mg zinc/kg-day. Chromosomal aberrations caused by zinc were observed in the bone marrow cells of mice maintained on a low calcium diet. It was thought that calcium may be displaced by zinc in calcium-depleted conditions, leading to chromosome breaks and/or interfering in the repair process (ATSDR, 1994).

Carcinogenicity

Studies of zinc exposure in humans have not specifically evaluated carcinogenicity. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk. The potential carcinogenicity of zinc has been evaluated in only a few animal studies. A summary of the currently available information is presented in IRIS (USEPA, 2000).

Occupational exposure studies to zinc dust or fumes have not reported an increase in the incidence of cancer, however, the studies were designed to evaluate other endpoints and did not specifically address cancer (USEPA, 2000). Epidemiological studies have examined cancer mortality rates in occupationally exposed workers and in residents in areas with potentially high zinc contamination. No association between cancer mortality and zinc exposure could be established for workers employed in electrolytic zinc and copper refining plants; however, analysis of the data was limited by the small number of deaths in workers exposed to zinc (Logue et al., 1982, cited in ATSDR, 1994). Lung cancer mortality was reported to be elevated in residents living in an old lead/zinc mining and smelting area, but there was no association with environmental levels of zinc (Neuberger and Hollowell, 1982, cited in ATSDR, 1994). Because many confounding factors (i.e., smoking, occupation, and duration of residence) were not considered, it is unlikely that the study could have detected zinc-related effects (ATSDR, 1994).

Newborn Chester Beatty stock mice were maintained for one year on drinking water containing 0, 1,000, or 5,000 ppm Zn (0, 170, 850 mg zinc/kg-day, as zinc sulfate), or on a diet containing zinc oleate (5,000 ppm Zn for 3 months followed by 2,500 ppm for 3 months, and then 1,250 ppm for the rest of the study period). The incidence of hepatomas, malignant lymphomas, and lung adenomas was not statistically different from control values, although the incidence of hepatomas in mice on the zinc-augmented diet was increased over

that in the controls (30.4 percent vs. 12.5 percent). In a 3-year, 5-generation study on tumor resistant and tumor-susceptible strains of mice, zinc concentrations of 10 to 20 mg/L in drinking water resulted in increased frequencies of tumors from the F_0 to the F_4 generation in the resistant strain (from 0.8 to 25.7 percent vs. 0.0004 percent in the controls), and higher tumor frequencies in two susceptible strains (43.4 percent and 32.4 percent vs. 15 percent in the controls). Statistical analysis of the data was not reported. Hypertrophy of the adrenal cortex and pancreatic islets, but no corresponding tumors were reported in C3H mice given drinking water containing 500 mg/L zinc sulfate for 14 months (studies cited in USEPA, 2000).

USEPA has given zinc a carcinogenicity weight-of-evidence classification of D, not classifiable as to human carcinogenicity, based on inadequate evidence in humans and laboratory animals.

Exposure Route Considerations

Ingestion

Zinc is essential for human beings, with the daily requirement recommended at 15 mg for adults and up to 19 mg for nursing mothers (NRC, 1979 as cited in ATSDR, 1994). Reports of toxic effects following ingestion of moderate amounts of zinc are uncommon due to an efficient homeostatic mechanism that regulates zinc levels in the body.

Zinc is usually present in tap water at concentrations below 0.2 mg/l although drinking water in galvanized pipes can contain up to 2 to 5 mg/l (NLM/HSDB, 1999). Typically, concentrations are much less than the secondary Maximum Contaminant Level (MCL) of 5 mg/L. This value is based on the threshold for metallic taste in water. Zinc levels in foods such as meat, fish, and poultry average 24.5 mg/kg, and grains and potatoes contain 8 and 6 mg zinc/kg, respectively. An estimate of daily intake of zinc for the adult U.S. population in food is 10 to 20 mg/day (ATSDR 1994).

Zinc interacts with other trace metals, and has a protective effect against toxicity from exposure to lead and cadmium (NAS, 1977). Excessive dietary zinc produces a copper deficiency in laboratory animals. Similar findings have been observed in humans receiving long-term treatment with zinc (ATSDR, 1994).

Inhalation

The Threshold Limit Values (TLV), 8-hour time weighted averages for zinc compounds in workroom air, are 1 mg/m³ for zinc chloride fumes and 5 mg/m³ for zinc oxide fumes (ACGIH 1999). Inhalation of elevated concentrations of zinc oxide fumes can produce metal fume fever. Zinc chloride particulate in air is more damaging than zinc oxide to the mucous membranes of the nasopharynx and respiratory tract upon contact, because zinc chloride is a corrosive (i.e. acid) salt.

Dermal

Occupational exposure to zinc oxide dust, combined with clogging of glands by dust, perspiration, and bacteria, has produced dermatitis (ATSDR, 1994).

Sensitive Populations and Indicators of Exposure

No specific data regarding human subpopulations that are unusually susceptible to the toxic effects of zinc were located (ATSDR, 1994). People who are malnourished or have a marginal copper status may be more susceptible to the effects of excessive zinc than people who are adequately nourished (Underwood 1977, cited in ATSDR, 1994).

Zinc is found in all human tissues and all body fluids and is essential for growth, development and reproduction. The total zinc content of the human body (70 kg) is about 2,300 mg (NLM/HSDB, 1999). Approximately 20 to 30 percent of ingested zinc is absorbed. It is principally excreted in the feces, though 300 to 600 µg/day is excreted in the urine (Goyer, 1991; ATSDR, 1994).

Toxicity Factors Derived for Risk Assessment

The oral reference dose (RfD) is based on a clinical study that investigated the effects of oral zinc supplements on copper and iron balance. A 10-week study of zinc supplementation in 18 healthy women given zinc gluconate supplements twice daily (50 mg zinc/day, or 1.0 mg/kg-day) resulted in a decrease of erythrocyte superoxide dismutase activity. There was a general decline in the mean serum high-density lipoprotein (HDL)-cholesterol in a higher-dose group (receiving 75 mg/day). USEPA (2000) reported that while it is not absolutely certain that the 50-mg zinc/day supplement (1.0 mg/kg-day) represents a clearly biologically significant endpoint, this level, when viewed collectively with other studies investigating effects on HDL-cholesterol, may signify the beginning of the dose-response trend. The significance of this change is unknown in light of an absence of increase in low-density lipoproteins (LDLs). The 1.0 mg/kg-day level was identified as LOAEL for zinc effects. An uncertainty factor of 3 was used, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient. The oral RfD for zinc is 0.3 mg/kg-day (USEPA, 2000).

A RfC or inhalation RfD has not been developed for zinc (USEPA, 2000).

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APPENDIX G

Sieved and Bulk EPA and USGS Samples

DRAFT FINAL SCREENING LEVEL HHRA
SPOKANE RIVER, WASHINGTON
Coeur d'Alene Basin RI/FS
RAC, EPA Region 10
Work Assignment No. 027-RI-CO-102Q

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APPENDIX G
Sieved and Bulk EPA and USGS Samples

This appendix contains scatterplot graphs comparing bulk and sieved concentrations of lead and arsenic in the USGS (Figures G-1 and G-2) and EPA (Figures G-3 and G-4) sampling data. Each graph shows the enrichment ratio of the sieved to bulk concentrations as estimated using linear regression techniques with the y-intercept of the regression forced through the origin (i.e., y = 0 when x = 0). Linear regressions are most commonly performed without forcing the y-intercept through zero, however, it was appropriate in this case due to the metal concentration relationship between bulk and fine-grained material. The y-intercept was forced through the origin because when the bulk metal concentration in a sample equals zero, the metal concentrations in both the 63 μm and 175 μm fractions of the sample, which are subsets of the bulk metal concentration, will also equal zero.

The enrichment ratio shown on the scatterplots is actually the slope of the regression line (the value in front of the "x" in the formula "y" = ratio multiplied by "x"). The term "x" is the bulk metal concentration and "y" is either the metal concentration in the 63 μm or 175 μm fraction. The scatterplots also show the r^2 (coefficient of determination) value. The r^2 value is a measure between −1 and +1 that indicates the proportion of the variation of the dependent variable (in this case, lead and arsenic concentrations in either the 63 μm or 175 μm fractions) that is explained by changes in the independent variable, which here is the bulk concentration of either lead or arsenic. An r^2 value close to +1 indicates a high positive correlation between the two variables (i.e., lead concentrations in the 63 μm fraction increases as bulk lead concentrations increase). An r^2 value close to −1 indicates a high negative correlation between the two variables (i.e., lead concentrations in the 63 μm fraction decrease as bulk lead concentrations increase. An r^2 value close to zero indicates that there is no relationship between the two variables.

A comparison of the analytical data for the 63 μm size fraction and the bulk sample for lead and arsenic shows an enrichment ratio of approximately two for both lead and arsenic. In other words, metal concentrations in the 63 μm size fraction are approximately double bulk concentrations. For lead, the high r^2 value of 0.8 indicates that the data are highly correlated, i.e., bulk concentrations and sieved concentrations are closely related. This conclusion is also shown in the graph where as bulk concentrations increase, sieved concentrations also increase in a linear fashion (Figure G-1). The lower r^2 value for arsenic of 0.4 (Figure G-2) indicates a weaker relationship between bulk and fine-grained fraction arsenic concentrations. The weaker relationship is depicted on the graph which shows more "scatter" in the data with not as

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SPOKANE RIVER, WASHINGTON
Coeur d'Alene Basin RI/FS
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consistent a pattern of increasing bulk arsenic concentrations related to increasing sieved arsenic concentrations (Figure G-2).

When lead concentrations in the 175 μm size fraction are compared with bulk concentrations, the enrichment ratio is slightly lower, but still lead in the 175 μm fraction concentrations approximately double that of bulk lead concentrations (Figure G-3). The r^2 value between the two fractions is also slightly lower, but still approximately 0.8. Therefore, the lead concentration increase in sieved samples relative to bulk samples is approximately the same whether concentrations from the 63 μm or the 175 μm size fractions are compared to bulk concentration data.

The enrichment ratio for arsenic in the 175 μm fraction relative to bulk concentrations is lower than the 63 μm to bulk enrichment ratio. Concentrations in the 175 μm fraction are approximately 1.5 times higher than bulk concentrations rather than two times higher as is the 63 μm to bulk concentration ratio for arsenic (Figure G-4). The r^2 between the 175 μm size fraction and bulk arsenic concentration is higher (approximately 0.8) than is the r^2 between the 63 μm size fraction and bulk data for arsenic, and there is correspondingly less "scatter" in the data depicted on the graph. This analysis indicates that the 63 μm size fraction appears to have arsenic concentrations approximately 50 percent higher than the 175 μm size fraction when compared to bulk concentration data. Bulk arsenic concentrations are a better predictor of arsenic in the 175 μm fraction than they are of arsenic in the 63 μm, as indicated by the higher coefficient of determination for the 175 μm fraction to bulk arsenic enrichment ratio. Therefore, using 63 μm concentration data in the lead model would not change the results of the risk analysis in the report – no additional sites would be selected for further action.

Figure G-1
Lead Concentrations for Bulk versus 63 um Sieved Samples (USGS Data)

$y = 2.1914x$

$R^2 = 0.8109$
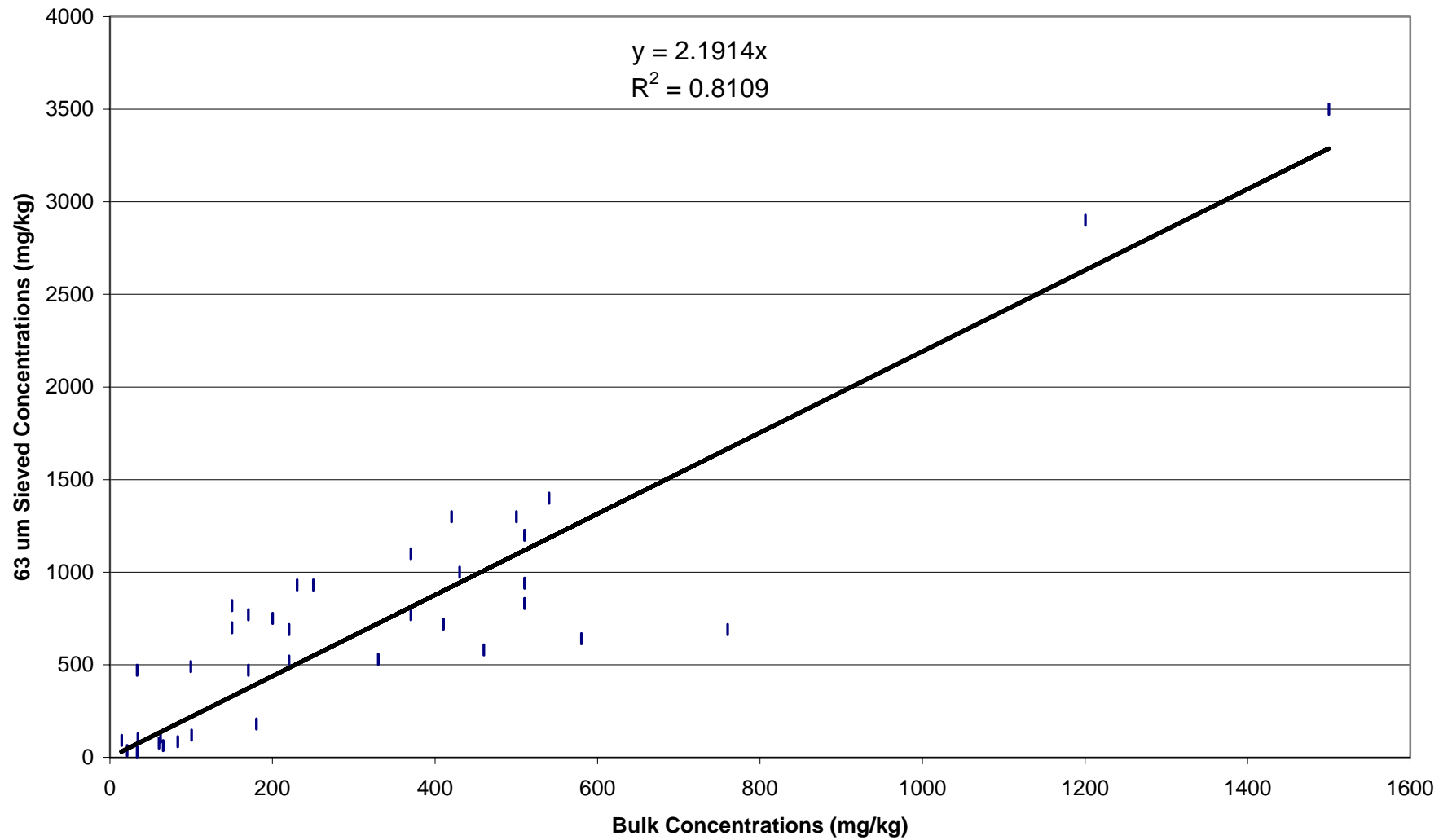
Figure G-2
Arsenic Concentrations for Bulk versus 63 um Sieved Samples (USGS Data)

$y = 1.9286x$
$R^2 = 0.3976$
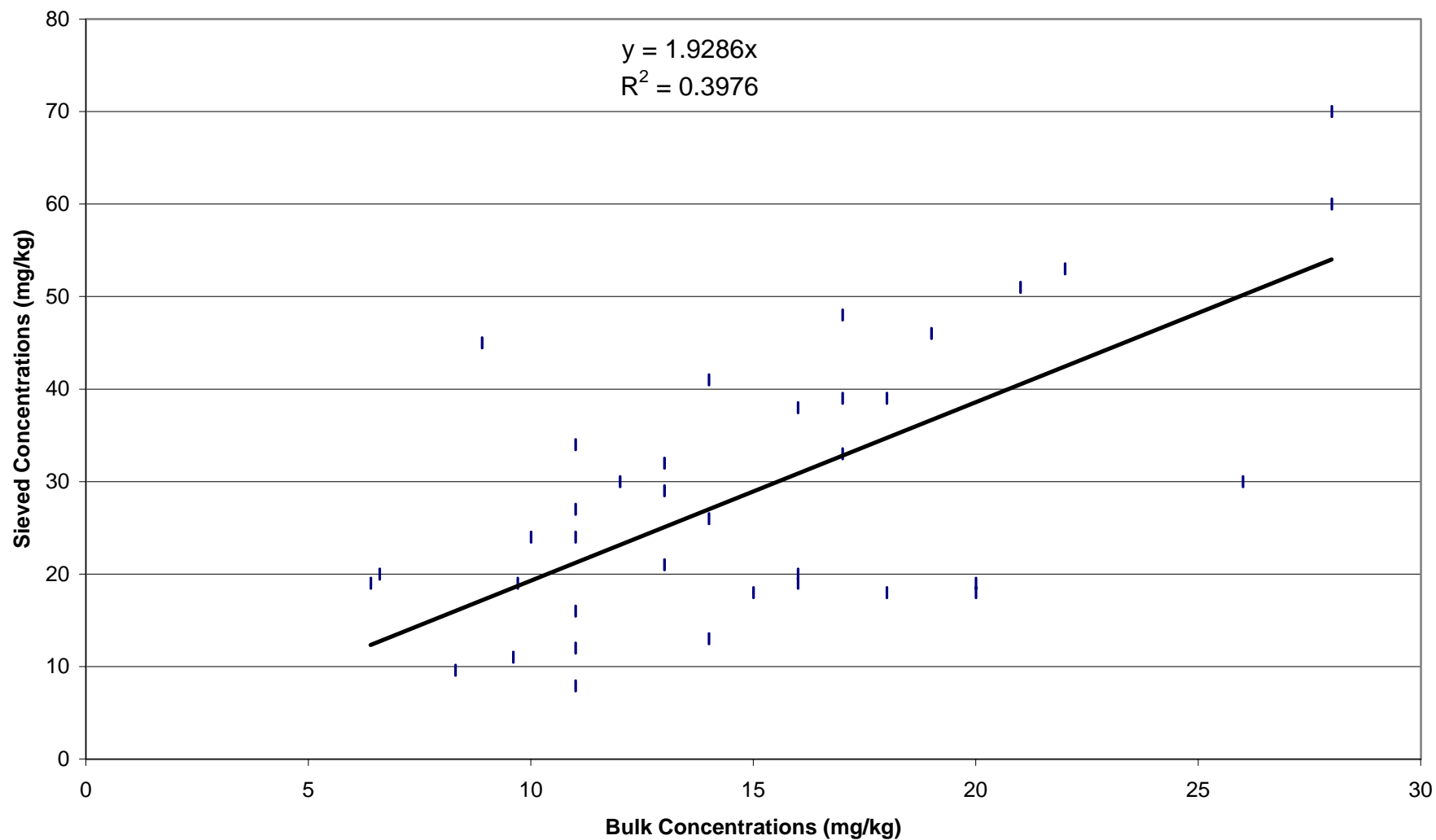
Figure G-3
Lead Concentrations for Bulk versus 175 um
Sieved Samples (EPA Data)

$y = 1.9585x$
$R^2 = 0.7639$
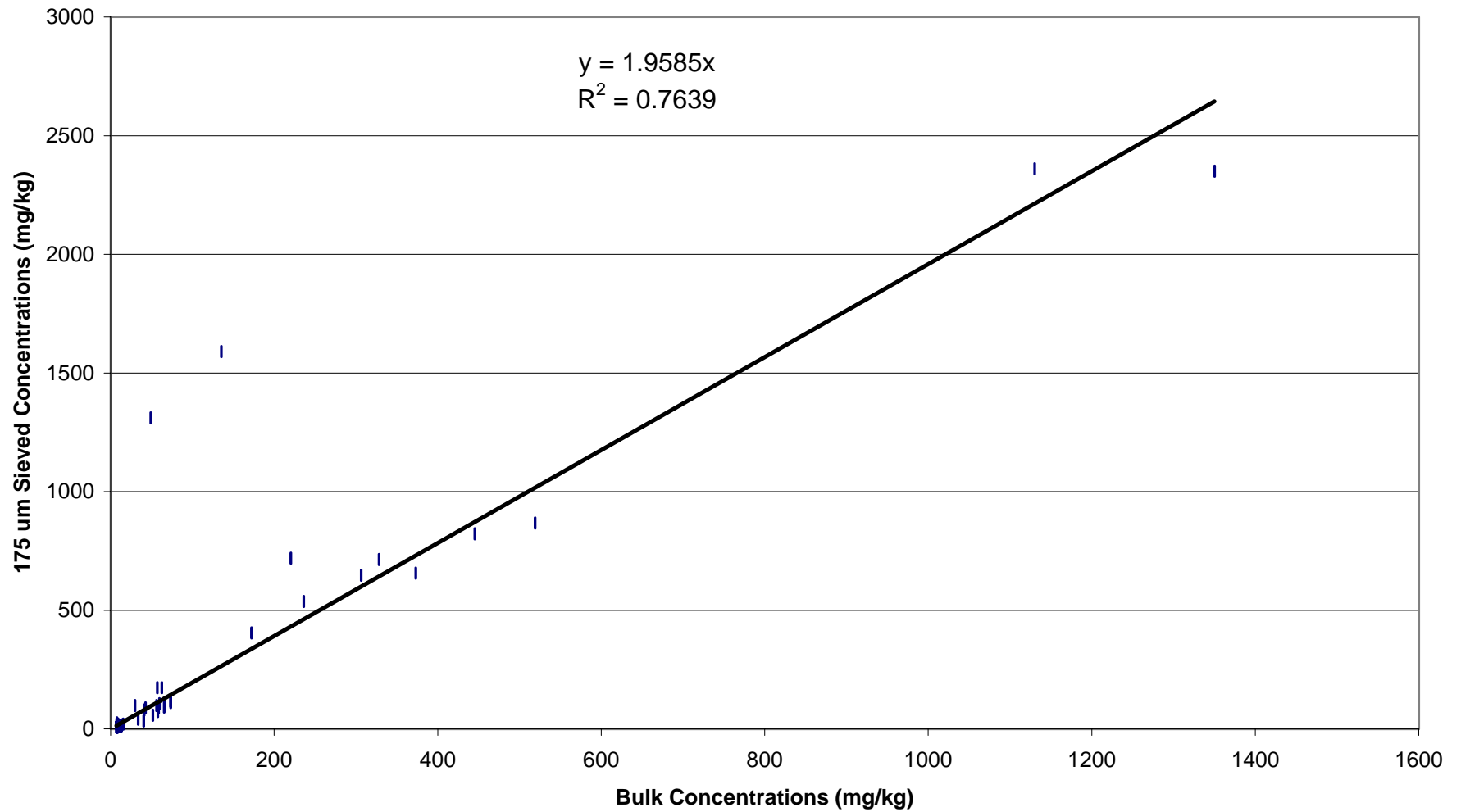
**Figure G-4
Arsenic Concentrations for Bulk versus 175 um
Sieved Samples (EPA Data)**

$$y = 1.5248x$$
$$R^2 = 0.7554$$

175 um Sieved Concentrations (mg/kg)

Bulk Concentrations (mg/kg)

APPENDIX H

UCL$_{95}$ Bar Graphs

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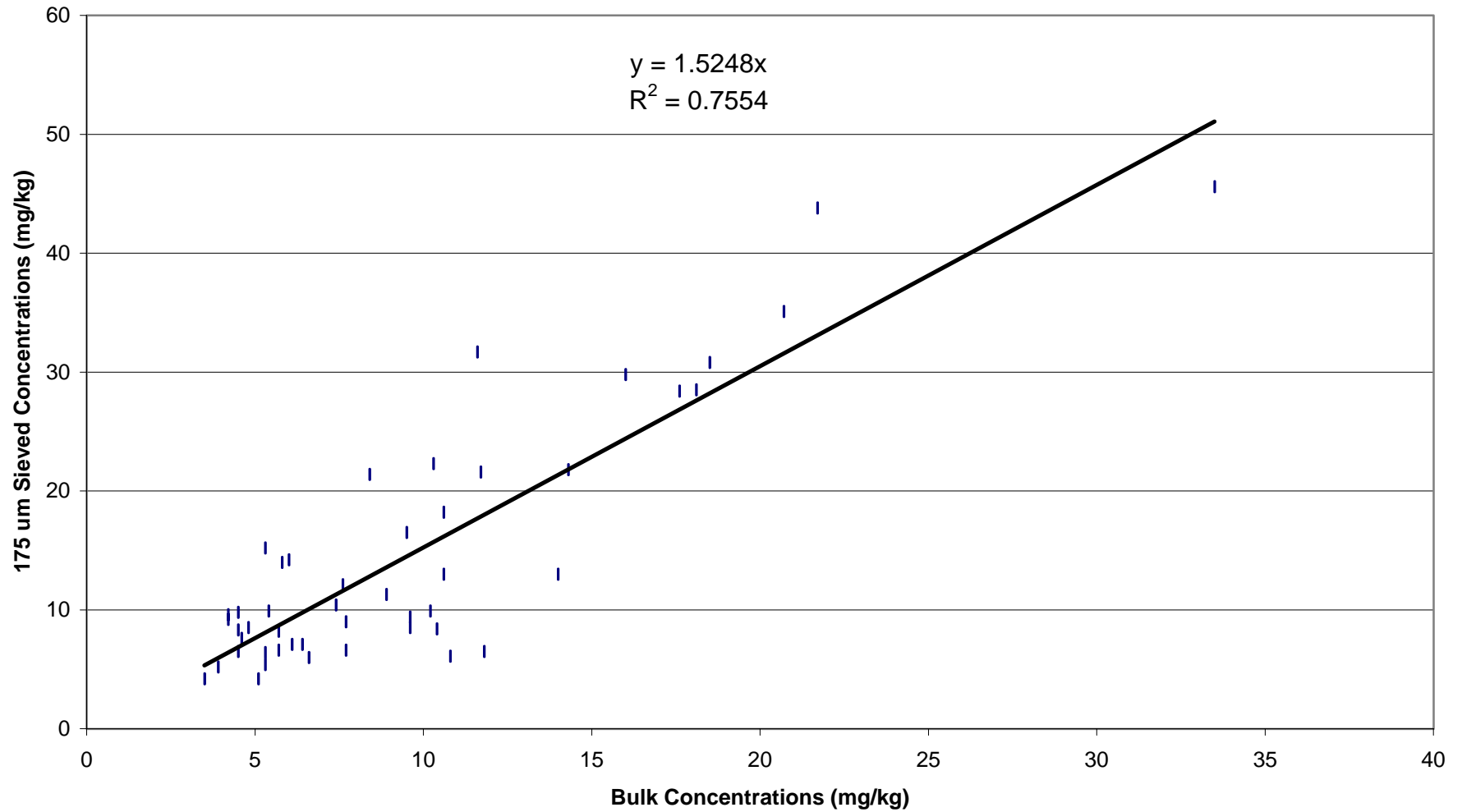
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APPENDIX H
UCL$_{95}$ Bar Graphs

The following figures illustrate the concentrations of the metals of concern at each of the common use areas. Generally, metal concentrations are highest upstream of Upriver Dam and decrease below.

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95% UCL Antimony Concentrations

Antimony (mg/kg)

Antimony Calculated
RBC 23 mg/kg

Spokane Area Background
Antimony Concentration Not
Available

Common Use Areas

201 - River Road 95
204 - Barker Road N.
208 - Boulder Beach(a)
217 - Myer's Landing(a)
220 - Jackson Cove(b)
223 - Horseshoe Point Campground(a)

Notes:
(a)-No 95% UCL was calculated at this site because antimony was not detected in any sample, therefore the detection limit was used.

DRAFT COEUR D'ALENE BASIN RI/FS
SCREENING LEVEL RISK ASSESSMENT, SPOKANE RIVER, WASHINGTON
RAC, USEPA Region 10
Work Assignment No. 027-RI-CO-102Q

Appendix H
Date: 6/9/00

95% UCL Arsenic Concentrations

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RAC USEPA Region 10
Work Assignment No. 027-RI-CO-102Q

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95% UCL Cadmium Concentrations

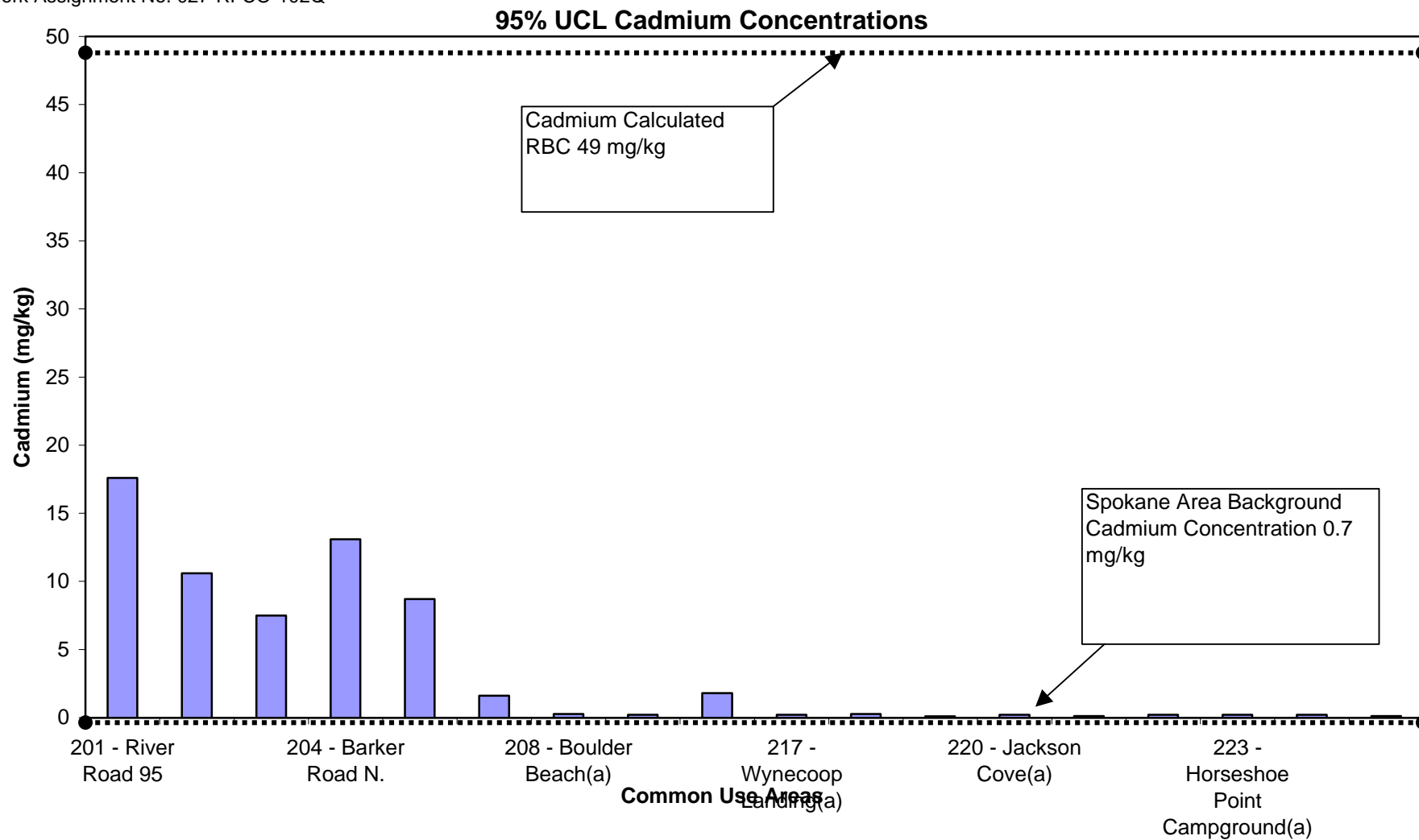Cadmium Calculated
RBC 49 mg/kg

Spokane Area Background
Cadmium Concentration 0.7
mg/kg

Cadmium (mg/kg) (y-axis, 0 to 50)

Common Use Areas (x-axis)

201 - River Road 95, 204 - Barker Road N., 208 - Boulder Beach(a), 217 - Wynecoop Landing(a), 220 - Jackson Cove(a), 223 - Horseshoe Point Campground(a)

Notes:
(a)-No 95% UCL calculated at this site because cadmium was not detected in any sample, therefore the detection limit was used.

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95% UCL Iron Concentrations

Spokane Area Background Iron Concentration 27,000 mg/kg

Iron Calculated RBC 17,109 mg/kg

Common Use Areas

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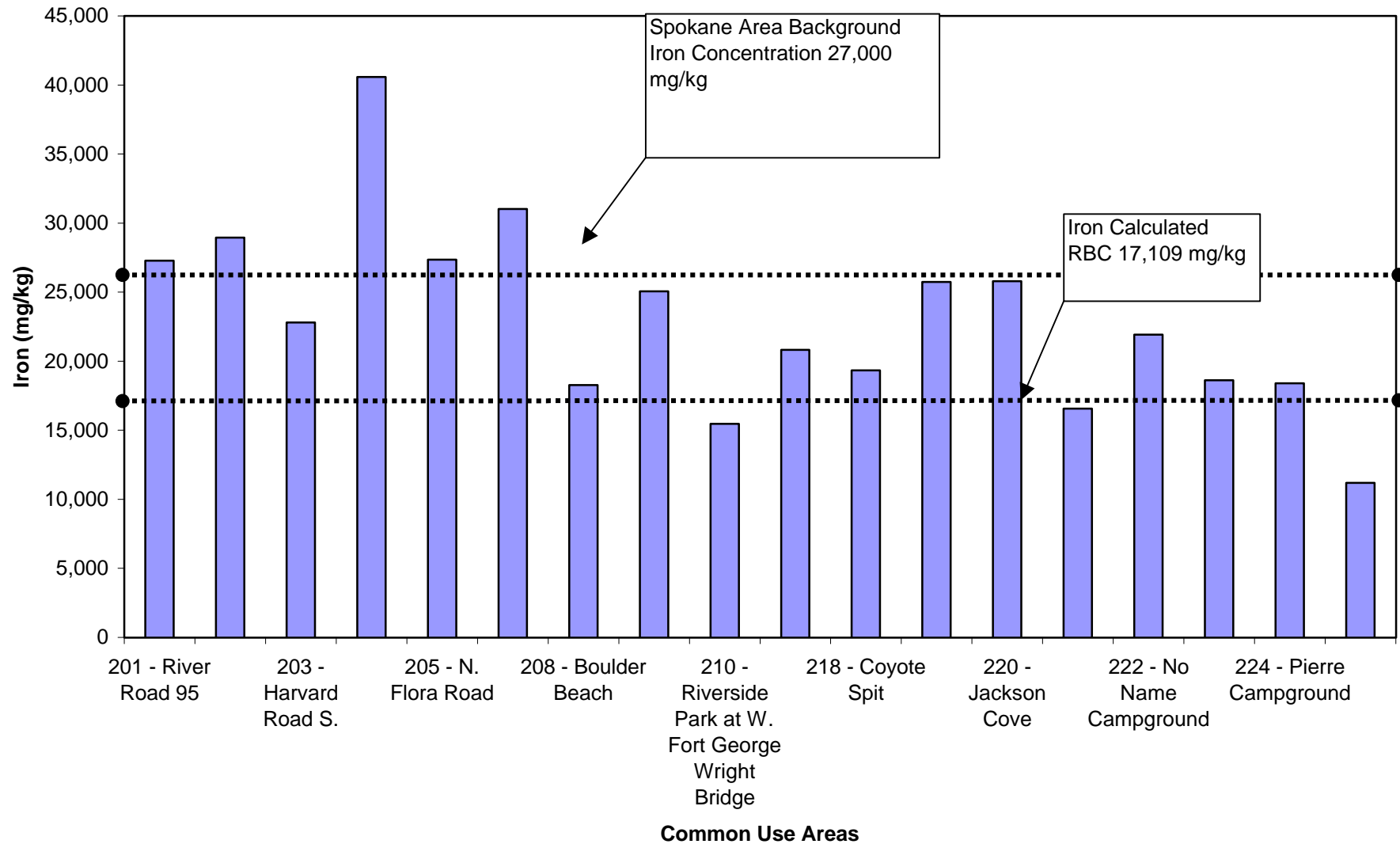
Average Lead Concentrations

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SCREENING LEVEL RISK ASSESSMENT, SPOKANE RIVER, WASHINGTON
RAC USEPA Region 10
Work Assignment No. 027-RI-CO-102Q

Appendix H
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95% UCL Manganese Concentrations

DRAFT COEUR D'ALENE BASIN RI/FS
SCREENING LEVEL RISK ASSESSMENT, SPOKANE RIVER, WAHINGTON
RAC, USEPA Region 10
Work Assignment No. 027-RI-CO-102Q

Appendix H
Date: 6/9/00
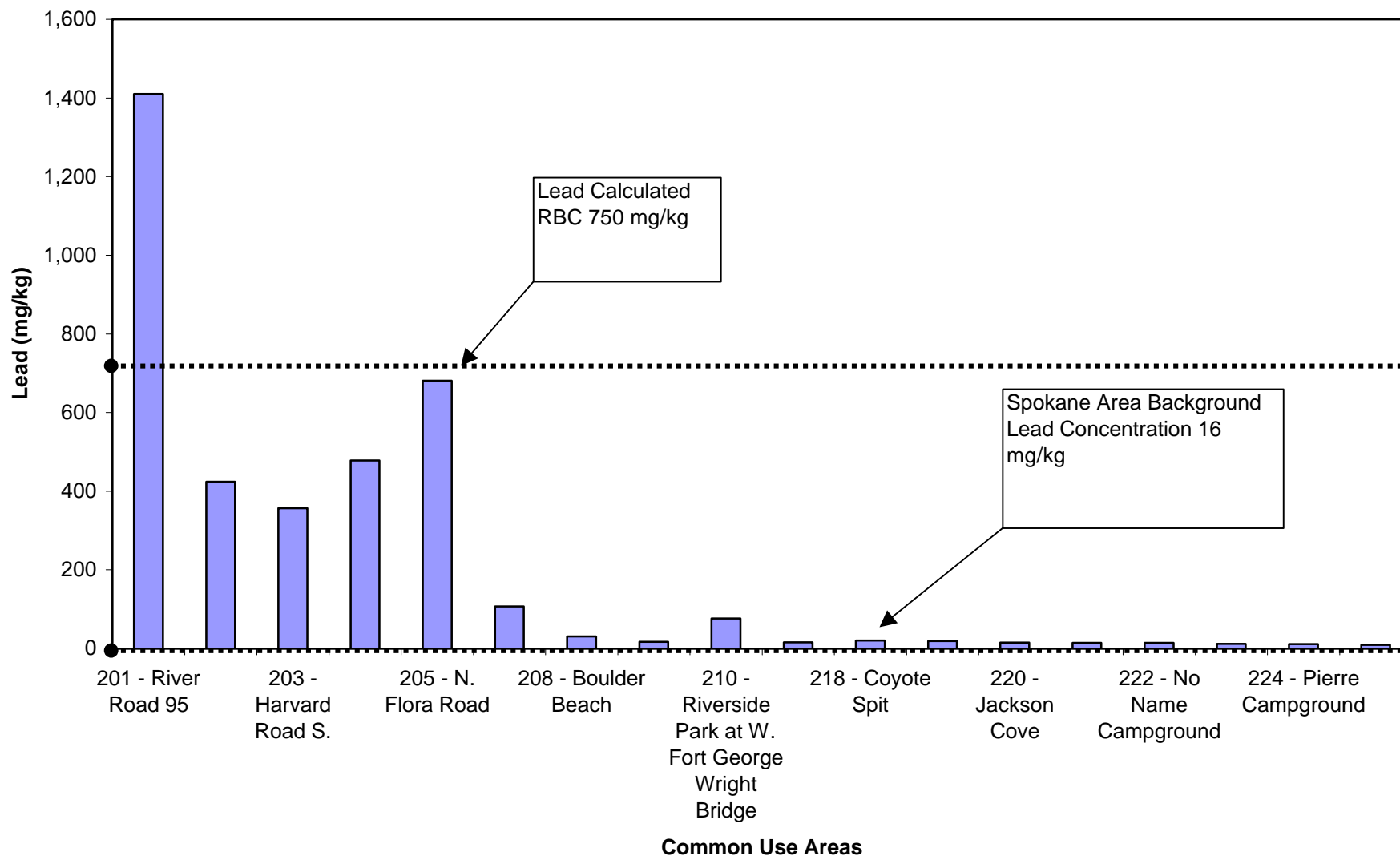
95% UCL Mercury Concentrations

Notes:
(a)-No 95% UCL was calculated at this site because mercury was not detected in any sample, therefore the detection limit was used.
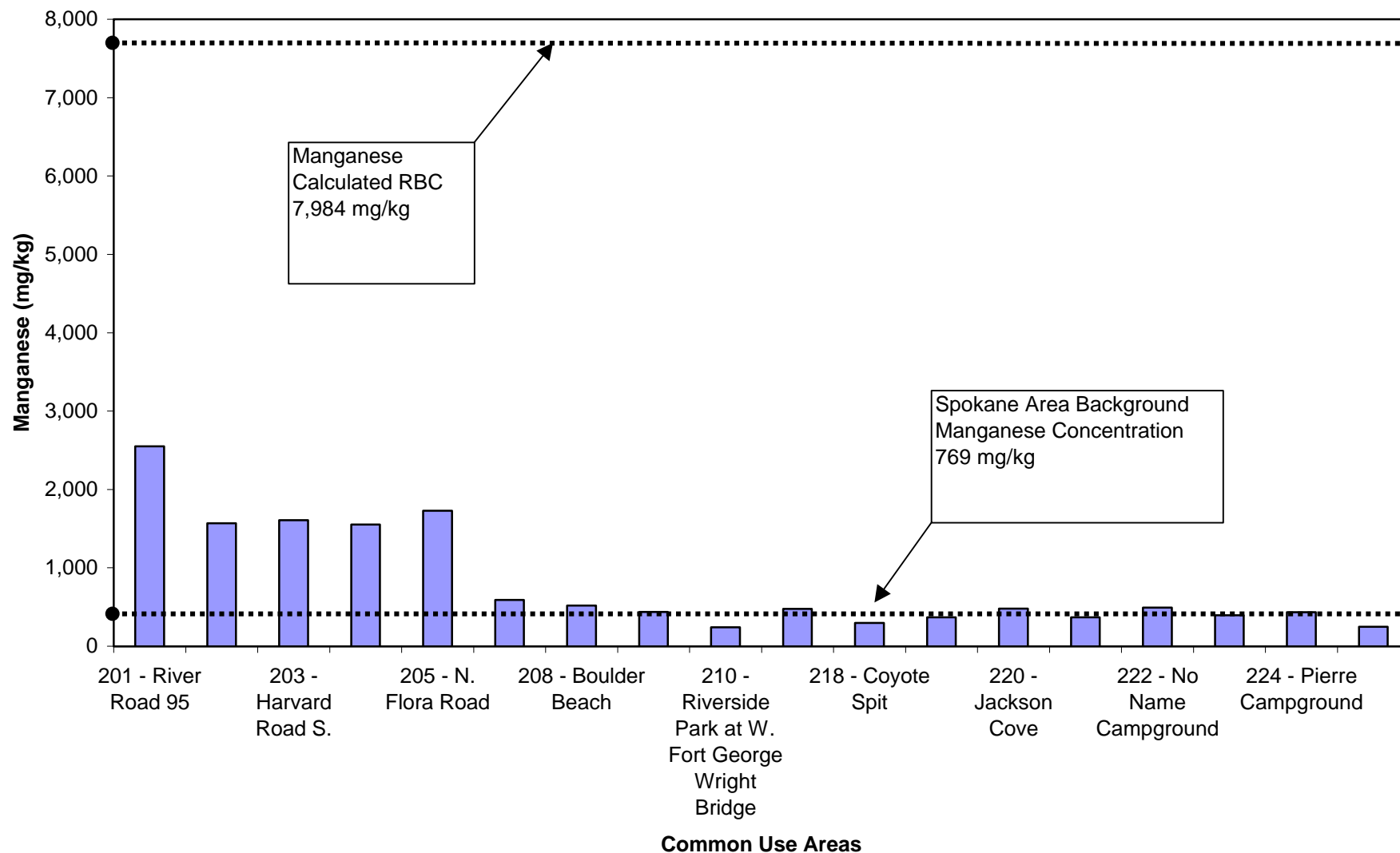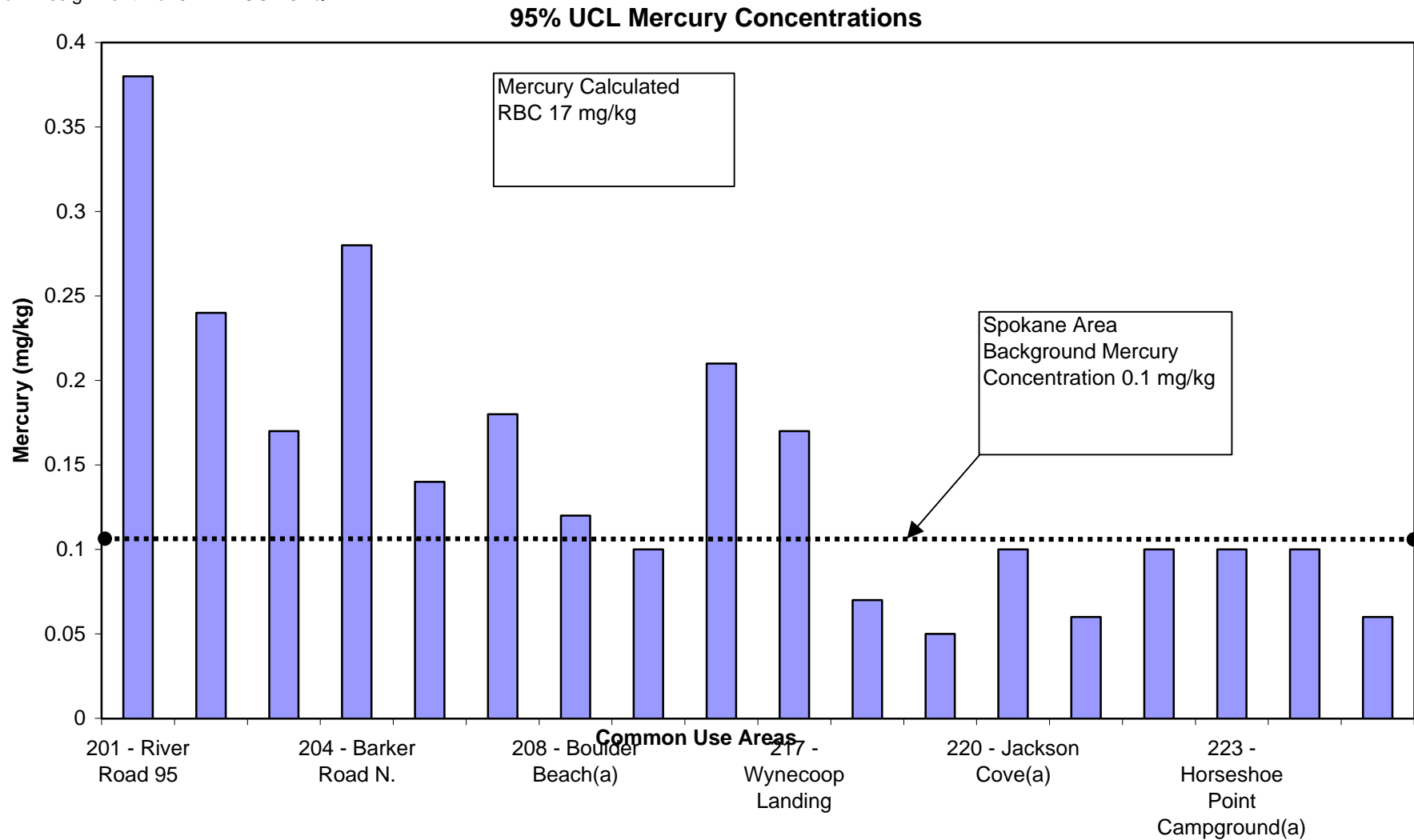
95% Zinc Concentrations

Chart showing Zinc (mg/kg) concentrations across Common Use Areas. Y-axis ranges from 0 to 20000.

Zinc Calculated RBC 17,109 mg/kg

Sp... Ba... Co...

X-axis categories (Common Use Areas): 201 - River Road 95, 203 - Harvard Road S., 205 - N. Flora Road, 208 - Boulder Beach, 210 - Riverside Park at W. Fort George Wright Bridge, 218 - Coyote Spit, 220 - Jackson Cove